snake venom poisoning

Findlay E. Russell, M.D., Ph.D.

Professor of Pharmacology and Toxicology
University of Arizona, Tucson, Arizona
Adjunct Professor of Neurology
University of Southern California, Los Angeles, California
Adjunct Professor of Neuropharmacology
Loma Linda University, Loma Linda, California

SCHOLIUM INTERNATIONAL, INC.

Great Neck, New York 11021

Printed and Published by:
Scholium International Inc.
265 Great Neck Road
Great Neck, New York 11021

Original Edition 1980
Reprint with Corrections 1983
Preface to Reprint Edition 1983

ISBN 0-87936-015-1

Library of Congress Catalog Card Number 83-3134

Printed in the United States of America

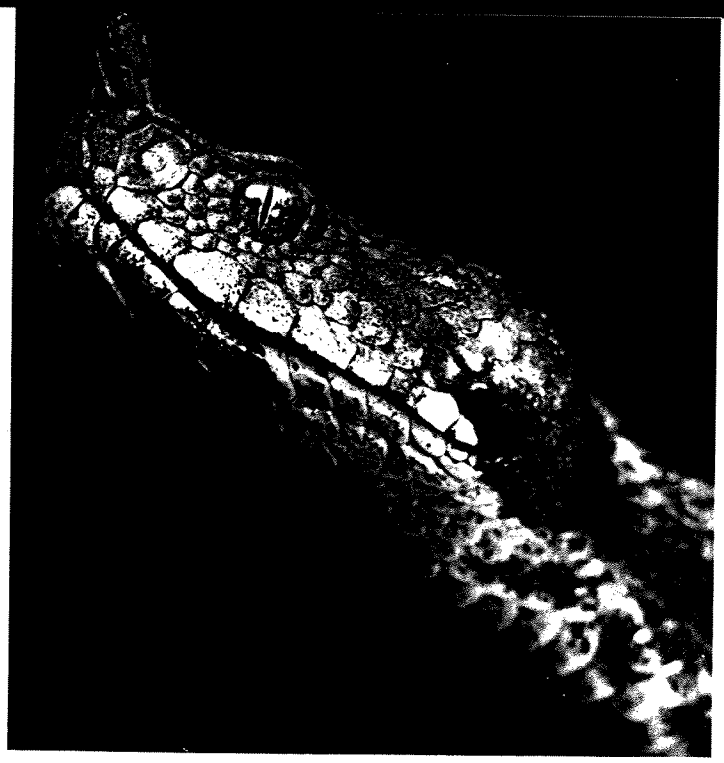
6 5 4 3 2 1

Library of Congress Cataloging in Publication Data
Russell, Findlay E.
Snake venom poisoning.

Includes bibliographical references and indexes.
1. Snake venom—Toxicology. 2. Poisonous snakes—North America. 3. Snakes—Mythology. I. Title.
RA1242.S53R87 1983 615.9'42 83-3134
ISBN 0-87936-015-1

8

exotic species in the United States



The physician confronted with a patient envenomated by an exotic snake may find himself bewildered.[1] Consultation is sometimes sought from the Communicable Disease Center in Atlanta, or from some other government office, none of which are organized to handle such emergency consultations. For reasons that seem difficult to understand, few physicians turn to the most obvious source—the local zoo—for immediate assistance.[2,3] Most major zoos have 24-hour services and will assist in contacting a consulting physician, as well as the nearest source of antivenin. The *Antivenin Index* in Oklahoma City (405-271-5454) maintains a 24-hour service on snakebites and is most proficient in handling advice on snakebite accidents. Another source of information is the Poisondex central office in Denver (800-332-3073). In some areas, the local poison control center is another source of information and provides consultation on bites by exotic snakes. Finally, some of the larger universities have herpetologists who are versed on exotic species and the treatment of their bites.

Bites by exotic snakes are becoming an increasingly important medical problem. Prior to 1950, there were few bites by nonnative species, but with the increasing collection of foreign snakes abroad, the importation of large numbers of reptiles by both amateur and professional herpetologists, an increase in the number of public exhibits of snakes, multiplied interests in

Table 8-1. Snakes Involved in 650 Cases of Snake Venom Poisoning

Family	*Genus*	*Common Name*	*Number of Cases*
Crotalidae	*Crotalus*	Rattlesnakes	483
	Agkistrodon	Cottonmouth, copperhead, and others	41
	Sistrurus	Pigmy rattlesnakes	10
	Bothrops	Fer-de-lance, and others	7
	Lachesis	Bushmaster	2
	Trimeresurus	Lance-headed vipers	6
Viperidae	*Vipera*	Russell's, European, and others	23
	Bitis	Puff adder, gaboon, and others	14
	Echis	Saw-scaled viper	4
	Cerastes	Horned viper	3
	Pseudocerastes persicus	Persian horned viper	1
	Eristicophis	McMahon's viper	1
Elapidae	*Micruroides*	Coral snake	8
	Micrurus	Coral snake	5
	Calliophis	Oriental coral snake	1
	Naja	Cobras	18
	Notechis	Tiger snake	2
	Demansia	Brown snake	1
	Oxyuranus	Taipan	1
	Dispholidus	Boomslang	2
	Bungarus	Krait	3
	Dendroaspis	Mamba	4
Hydrophidae	*Laticauda*	Yellow-lip sea snake	3
	Enhydrina	Beaked sea snake	2

venomous snakes and snake venoms by scientists, and the shenanigans of exhibitionists, there has been an impressive increase in the number of exotic snake envenomations during the past 15 years. Although the number of exotic snakes in the United States today is unknown, a study in Southern California showed there were 1,875 of these snakes in collections in that area.* During 1970 to 1971, 75,223 snakes, including 6,836 venomous species, were imported, mostly for the pet trade.[4] This number may not be realistic, because a good many snakes are smuggled into our country. In the past 4 years, I have treated one customs official and one wildlife worker who inadvertently reached into a box labeled "Biological Material—Handle with Care," only to discover that the biology was reptilian, and alive.

A mail survey of 10 herpetologists or snake collectors and handlers in Southern California indicated that they kept a total of 667 nonnative venomous snakes. They suggested that there may be as many as 2000 exotic snakes in the area. Between 1970 and 1977, our facility held a total of 47 exotic snakes confiscated by customs officials, California Fish and Game, and various city, county and state animal control offices.

In our own practice, we had attended 20 bites by nonnative species by the year 1968.[5] This number represented approximately 10 per cent of the total number of snakebites. By 1972, this percentage had risen to 12. The 62 cases, reported in 1972, included not only patients seen at medical facilities in the far west, but patients treated by the author in various other parts of the world.[6] By the year 1978, we had treated 85, or 13 per cent of our patients, for bites by nonnative species (see Table 8-1). It should be noted that the Los Angeles County-University of Southern California Medical Center has specialized in the handling of exotic snake venom poisonings over the past 30 years, and this high incidence is not typical of experiences elsewhere in the country.

Parrish noted that in one series of cases he reviewed, approximately 4 per cent of the patients were bitten by exotic snakes.[7] Three of the six snakebites treated at the Roosevelt Hospital in New York City between 1965 and 1972 involved exotic species,[8] while Minton noted that the Bronx Zoo supplies antivenin for bites by two nonnative species each year for the New York City area.[9]

Between 1955 and 1977, we logged a total of 373 telephone calls and 121 letters relating to bites by exotic snakes. Of the telephone calls, approximately 50 involved patients bitten by an exotic species. Between1966 and 1976, we were consulted on 18 envenomations by exotic snakes.

It is not possible within the scope of this text to discuss all of the venomous snakes of the world or the bites they inflict. But since there are many thousands of exotic snakes currently in United States zoos and collections, some mention is made of several of the more medically important of these snakes.

*Strassberg, J.: Personal communication, 1963.

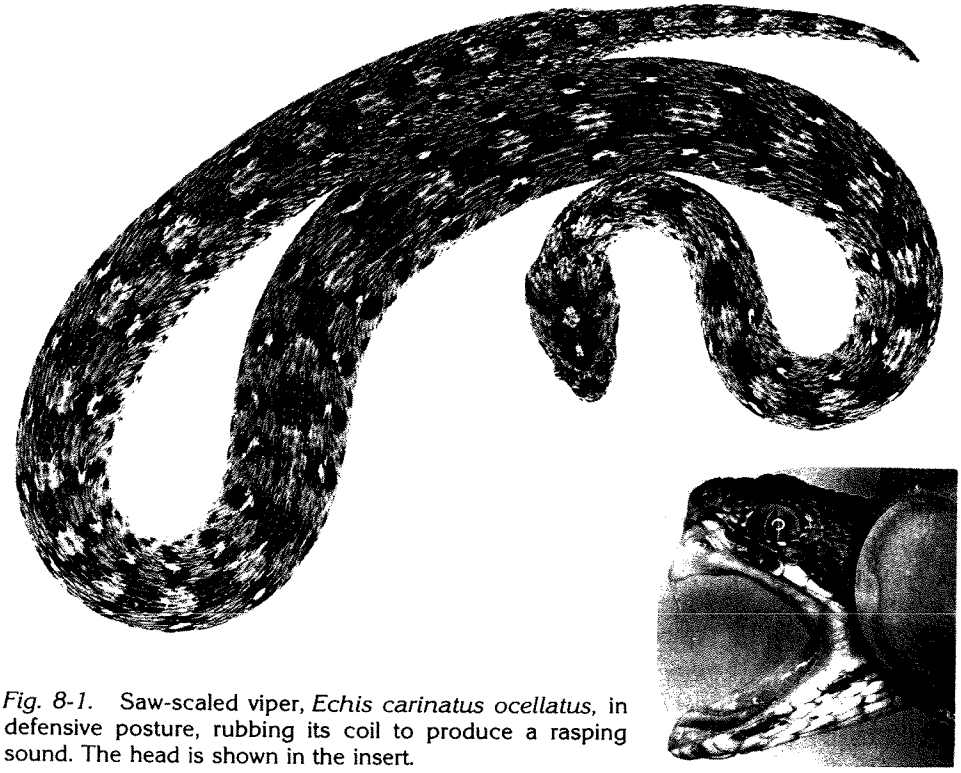


Fig. 8-1. Saw-scaled viper, *Echis carinatus ocellatus,* in defensive posture, rubbing its coil to produce a rasping sound. The head is shown in the insert.

Fig. 8-2. Swelling and persistent bleeding occur from incisions made at area of bite.[10]

There is also the possibility that someone might release a foreign snake in the wild. This is more than a theoretical possibility: In1962, I was called by an officer in Orange County who had reported the capture of a Russell's viper on the desert near Indio. At first it was reported that someone had been bitten by this snake, and I was asked to identify it. Thinking there had been a

misidentification, I asked for the snake to be sent to the Los Angeles County General Hospital. The snake was very definitely a Russell's viper, and I immediately asked for more information about the reported bite. Apparently, envenomation had not occurred, and only a strike took place when a man reached underneath an outdoor water heater to relight the pilot. He was struck on the hand by the viper, which had curled up beneath the tank. Further investigation indicated that a Russell's viper had been stolen the previous week from a pet store approximately 60 miles away. While we may never know exactly what happened, I can imagine the proud bandit, and erstwhile owner of this Asian viper, thumbing through his book on snakes. Discovering he was the new owner of a very dangerous snake, and being a good conservationist, he immediately dispatched the reptile to the nearest desert.

The reader will find more data on exotic snakes and their antivenins in Chapter 7. On the basis of collections in nine of our major zoos, and data provided me by the *Antivenin Index,* I have selected eight of the most common foreign snakes currently housed in this country for review. All of these have been implicated in bites on humans within the United States during the past 25 years.

Saw-Scaled Viper
(Echis carinatus)

According to Warrell, "this snake probably bites more people than any other species of snake,"[10] over the same range—Senegal to Bengal.[10] The adult of this species is a moderately stout snake, 14 to 20 inches (35–50 cm.) in length, and from pale buff or tan to olive brown, chestnut, or even reddish in color, and often varying considerably in color throughout its distribution (Fig. 8-1). A light trident or arrowhead marking against a brown background is often seen on the dorsum of the head. When stimulated, this rather hypertonic snake rubs its inflated coils together and produces a sizzling or rasping sound, which has given rise to many native names for the snake.

SYMPTOMS AND SIGNS OF ENVENOMATION

The symptoms and signs of poisoning are pain, often immediate, and sometimes radiating from the bite area up the arm or leg; local swelling, with ecchymosis and bleb formation (Fig. 8-2); and in some cases, necrosis. Of Warrell's series, 93 per cent had incoagulable blood.[10] Spontaneous bleeding and disseminated intravascular coagulation is seen in all but minimally envenomated patients. Spontaneous bleeding occurs from the gums, nose and other mucous membranes (Fig. 8-3). Warrell found fibrinogen severely depleted, and fibrin degradation products increased, but significant thrombocytopenia was seen in less than 10 per cent of the serious cases.[10] Reid also

commented on this.[11] Nausea, vomiting, drowsiness, and headaches are less frequently reported. Central nervous system hemorrhage may give rise to convulsions and shock. Spontaneous bleeding was the cause of death in 5 of 115 patients studied by Warrell.[10]

TREATMENT OF ENVENOMATION

Treatment includes the immediate injection of antivenin. In Warrell's series, mortality was reduced from a range of 10 to 20 per cent to 2.8 per cent through the use of antivenin.[10] In the four patients treated or seen by the author, Behringwerke or Institut Pasteur antivenins were employed. In one case, eight vials of antivenin were injected intravenously following the almost immediate appearance of edema and oozing of blood from the gingival sulcus. Warrell noted in his cases, "the sole indication for the use of antivenin was incoagulable blood indicating systemic poisoning."[10] Since the simple clotting test that he used to diagnose systemic poisoning can be carried out at 20 minutes, this seems good advice. Unfortunately, we were not aware of this procedure when we saw our cases of *Echis carinatus* envenomation. The rapid onset of swelling and bleeding from the gums prompted us to give antivenin in this one case, and the patient had an uneventful recovery. His clotting screen showed definite abnormalities, but since blood was taken after antivenin had been started, we had difficulty in evaluating the significance of the findings.

In one other patient, three vials of antivenin were given after the onset of pain and edema. There were no bleeding phenomena, and the clotting screen was normal. The patient had an uneventful recovery. In the remaining two cases, there was little evidence of envenomation and no antivenin was administered. We have not used the South African Institute of Medical Research antiserum. Warrell noted that he observed significantly more immediate reactions following the use of this antivenin than with use of Behringwerke or Institute Pasteur materials. These reactions resembled pyrogen-like responses. Delayed serum reactions were rare in Warrell's large series of cases.[12]

Transfusions and fluid replacement are essential, and a program similar to that outlined for rattlesnake bites should be followed. Codeine phosphate is suggested for pain. Aspirin should be avoided, since the venom of this snake produces disturbing alterations in bleeding and blood coagulation. The appropriate anti-tetanus agent should be given.

Puff Adder
(Bitis arietans)

The adult puff adder is 3 to 4 feet (90–120 cm.) in length, and a few reach 6 feet (1.8 meters; Fig. 8-4). This snake may strike with astonishing rapidity

Fig. 8-3. Spontaneous bleeding from gingival sulcus occurs following a bite by *Echis carinatus.*[10]

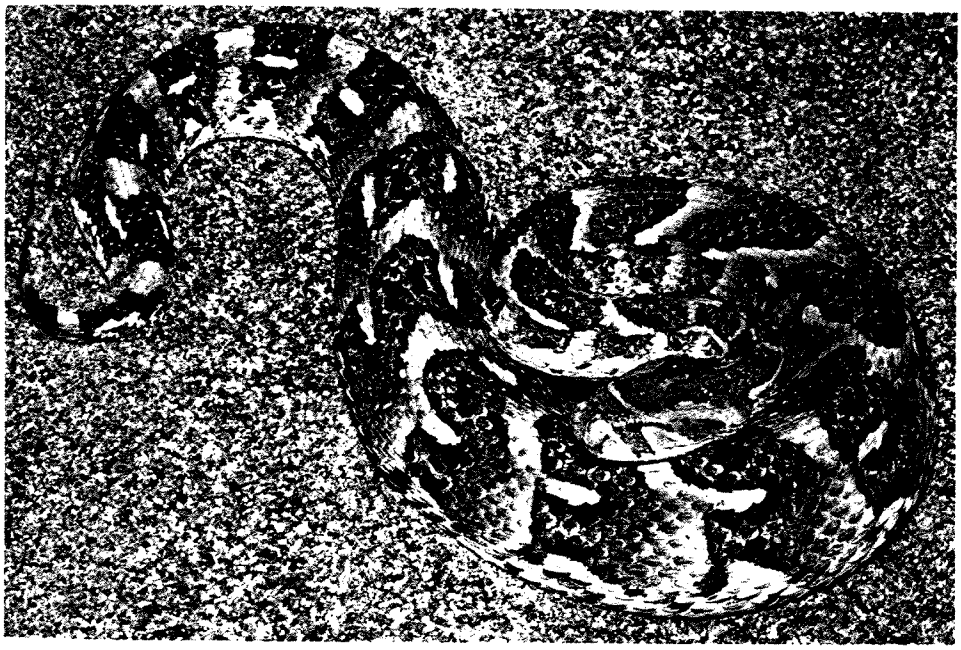


Fig. 8-4. Puff adder, *Bitis arietans.*

and accuracy. It is particularly dangerous to handle, because of its nasty habit of giving a quick jerk when it is being held.

SYMPTOMS AND SIGNS OF ENVENOMATION

The symptoms and signs of puff adder bite are very similar to those of rattlesnake bites: pain, swelling, ecchymosis, bleb formation, lymphadenitis, and lymphangitis (Figs. 8-5, 6). Tissue necrosis may develop, and nausea and vomiting have been reported, as has thrombophlebitis. In severe poisoning, shock may occur. One patient treated by the author developed hematuria 16 hours following the bite.

Fig. 8-5. Bite by puff adder, showing swelling, ecchymosis, and bleb formation 2 days after bite.

Fig. 8-6. Bite by puff adder, showing bleb formation with extensive extravasation.[13]

TREATMENT OF ENVENOMATION

Treatment includes the immediate intravenous administration of antivenin. We have used the South African Institute of Medical Research polyvalent antivenin in all four of our cases. In each, the antivenin was given within 90 minutes of the bite. A minimum of 50 ml. should be injected. Fluid replacement in the form of albumin or whole blood may be needed in all severe envenomations, but saline or glucose solutions are adequate in most cases. Further therapeutic procedures should follow those suggested for rattlesnake bites.

Of 210 cases reported by Visser and Chapman, 57 (27%) were classified as severe poisonings. Of these 57 severe envenomations, 29 (52%) died.[13] Only one of our cases could be considered a severe envenomation.

Black Mamba
(Dendroaspis polylepis)

The adult black mamba is a relatively long (6–14 feet, or 1.8–4.2 meters), slender snake, varying in color from gray to uniformly or blotched brown, or grayish brown (Fig. 8-7). It is never coal black but may appear a dark, olive green, particularly before shedding. The inside of the mouth is black, which easily differentiates it from other venomous snakes in Africa. The snake can strike so quickly that the victim may be unaware he has been bitten.[13] Its length provides a large specimen with the ability to strike a distance of 5 or 6 feet (1.5–1.8 meters), and it can strike above the belt. It may deliver several quick bites when threatened. Of all the snakes, I consider this the most dangerous to handle, not only because of its inherent speed, but because of the potency of its venom. The lethality of the venom is demonstrated by a 100-per-cent mortality in the seven cases reported by Visser and Chapman.[13]

SYMPTOMS AND SIGNS OF ENVENOMATION

The symptoms and signs of envenomation may include early dyspnea and a feeling of tightness in the throat, dysphagia, slurred speech, and muscle spasms and fasciculations, followed by marked weakness or paralysis, respiratory difficulty, and increased salivation. The pulse may be normal or increased, while blood pressure may be normal at first but then falls to shock levels in severe poisoning. There may be some nausea and vomiting, and ptosis, but pain and swelling are usually minimal (Fig. 8-8).

TREATMENT OF ENVENOMATION

Treatment includes immediate intravenous administration of antivenin. In the single case seen by us, six vials of the mamba trivalent antivenin were given intravenously, with good results. This antiserum is no longer available, and we advise the South African Institute for Medical Research polyvalent antivenin. We have not used the Behringwerke "Central Africa" polyvalent serum but have had good reports on its efficacy. Oxygen should be administered if it is not contraindicated, and the patient should be observed closely for respiratory difficulties, and obstruction by pharyngeal fluids. If respiratory changes do occur, pharyngeal drainage should be instituted immediately, and a tracheostomy considered. The routine treatment for shock should be instituted if blood pressure begins to fall. Other symptoms and signs are treated symptomatically. The appropriate anti-tetanus injection should be given.

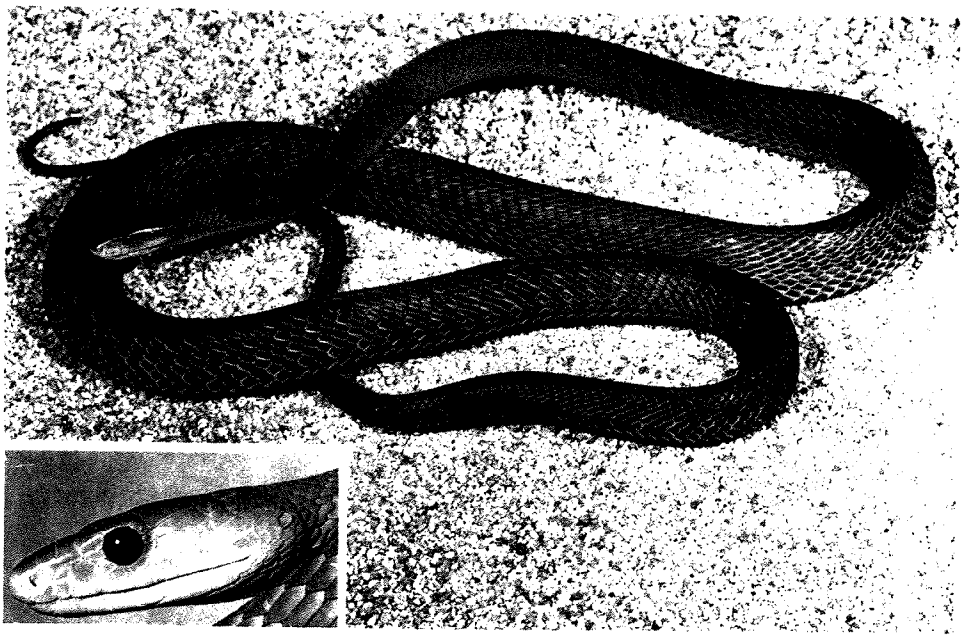


Fig. 8-7. Black mamba, *Dendroaspis polylepis.* The head of a mamba is shown in the insert.

Fig. 8-8. Bite by black mamba. In this patient, a vesicle developed at the site of the bite, and there was some erythema, and mild swelling.

We have performed renal dialysis in several dogs exposed to lethal doses of this venom. The procedure was initiated within 10 minutes of the venom injection. Both dogs survived and were eating 24 hours later. Visser and Chapman advise renal dialysis in the treatment of renal failure.[13]

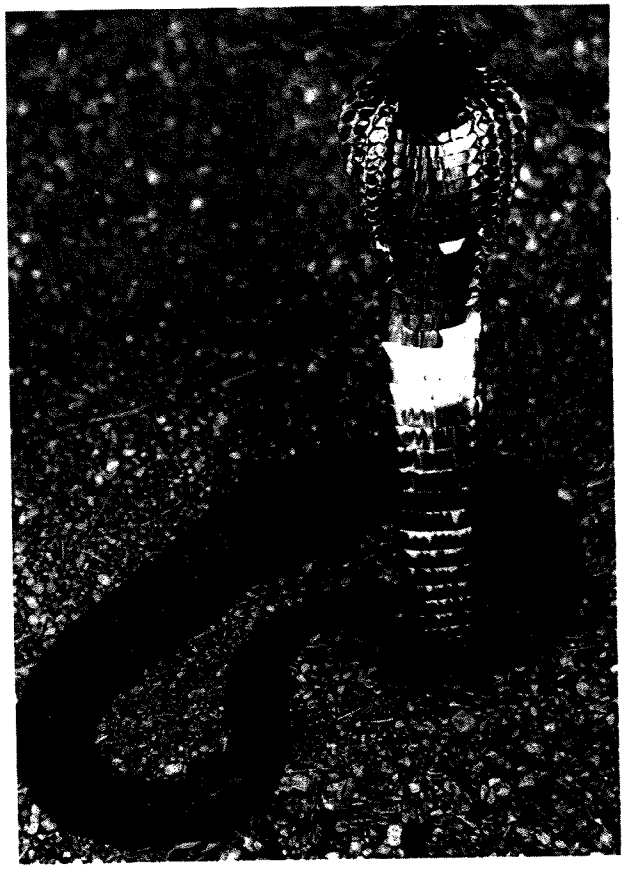


Fig. 8-9. The ringhals, *Hemachatus haemachatus,* a spitting cobra, is pictured in its normal posture prior to ejection of venom.

Spitting Cobra
(Naja nigricollis)

"Spitting" cobras are elapids whose fang structure and fang discharge orifice permits them to "spit," "spray," or eject venom as a defensive act. These modifications in the fangs allow the venom to be ejected forward in a single jet from each fang, rather than downward, as it is in most other snakes. By raising the head and the forepart of its body from the ground, and then tilting its mouth beyond a plane horizontal to the ground, these cobras can eject a stream of venom from one or both fangs through their slightly parted jaws. When spitting, *Hemachatus* assumes a more erect position than either *Naja nigricollis* or *N. mossambica* (Fig. 8-9). It does not need to tilt its jaws. However, both snakes can spit with equal proficiency from the ground without

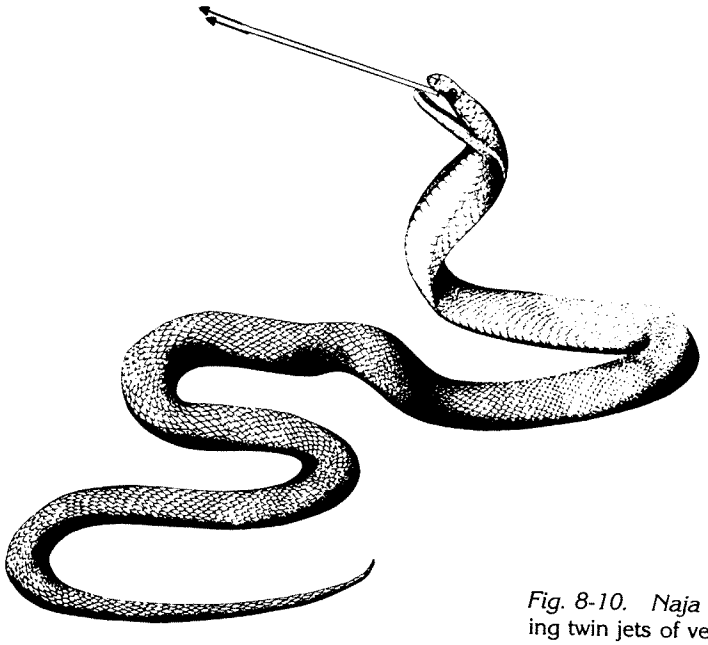


Fig. 8-10. Naja nigricollis ejecting twin jets of venom.

raising their heads.[14] It is interesting to note that non-spitters do not direct the head upward when approached by humans, as Joan Root pointed out to Charles Bogert in 1978.* The spitting cobras direct their venom with considerable accuracy by coordinating their head and neck movements. Figure 8-10 shows the posture of *N. nigricollis* when spitting venom. This drawing is based upon a photograph of a cobra spitting at an assistant of Mr. Henry C. Raven at Kafue, northern Rhodesia.

There are many reports and numerous field observations on the spitting of the cobras.[15-19] Ditmars describes the phenomenon as follows:[20]

> . . . the performance is accomplished with the jaws slightly parted. . . . The performance is very quick. . . . The snake rears and it may instantly spring to the pose. Facing the object . . . it looks intently into one's face. . . . If it seeks to direct the poison upwards it curves its rearing pose backward, thus directing the head upwards. The ejection of the poison is an instantaneous operation. The jaws are slightly opened and closed so quickly as to appear like a snapping motion and during this action the poison leaves the fangs. There is no dribbling or spilling of the fluid. It issues in twin jets and the jaws of the snake are clear of it when the feat is accomplished. There is every indication that, at the instant the snake prepares to eject the poison, it contracts the temporal (capti-mandibularis superficialis-?) muscle over each gland, thus producing pressure to force the toxic fluid a considerable distance. This flies with such force that its impact can be distinctly heard against ordinary glass five feet away. At the instant of the ejection the snake emits a sharp hiss. This ejection of air might be an accompanying token of anger, or it may assist the travel of the poison.

*Bogert, C. M.: Personal communication, 1978.

As noted by Bogert, there are a number of cobras that can qualify as spitters. Several subspecies of the Asiatic cobra, *Naja naja,* including those in East India, Burma, the Malay Peninsula, Java, and the Philippine Islands, have been reported to spit under some conditions. West of Bengal, however, there do not appear to be any authenticated reports of cobras ejecting their venom during a spitting act.[19] The king cobra, *Ophiophagus hannah,* is not known to spit, and its fangs are not modified to eject venom. In Africa, almost every species of cobra has been reported as a spitter, but without doubt the episodes reported involved a misidentification: the real culprit being *N. nigricollis, N. mossambica,* or *Hemachatus.* Other than some subspecies of *N. naja,* two of the five species in Africa *(N. nigricollis* and *N. mossambica),* and *Hemachatus haemachatus* (cobras with a discharge orifice on the fang that has been modified for the forward ejection of venom), the spitting talents of other *Naja* species must be considered questionable, although the sudden exhalatation of air that some cobras exercise when ejecting venom may propel the venom a short distance.

The spitting cobras can be differentiated from the non-spitting cobras by several characteristics, as shown in Figures 8-11 and 8-12. In the former snakes, the suture or weld of the fang is on the median line of the anterior surface of the curve. The discharge orifice is located toward the center of the fang at the end of the suture, and it is smaller and of a different shape than that in the non-spitting cobras. In the latter, the venom canal continues as an open groove extending almost to the point. Bogert considers the ringhals *(Hemachatus)* fangs as the most perfectly adapted for spitting.[19]

Reports on the distances that these cobras can spit are subject to as much variation as the lengths of rattlesnakes. Rose stated that the venom may be ejected 6 or 7 feet,[21] while Ditmars[20] and Fitzsimons[14] both reported a figure

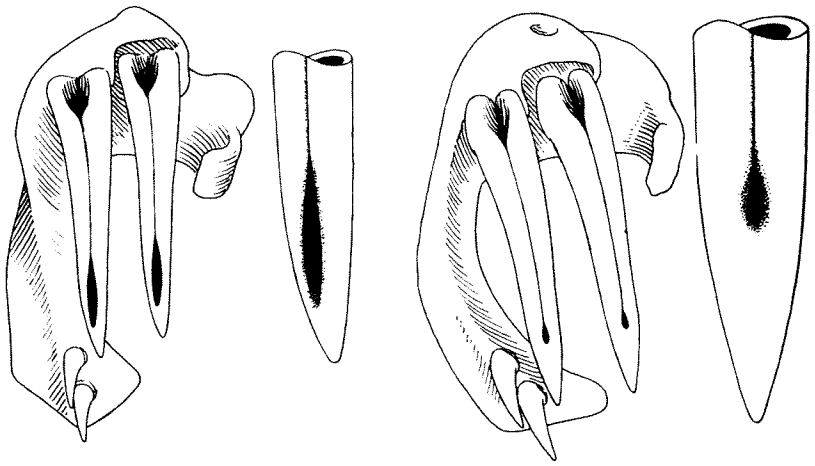


Fig. 8-11. A ventral view shows the suture and discharge orifice of the non-spitter, *Naja haje anchietae* (*left*) and the spitter, *Naja nigricollis* (*right*).[19]

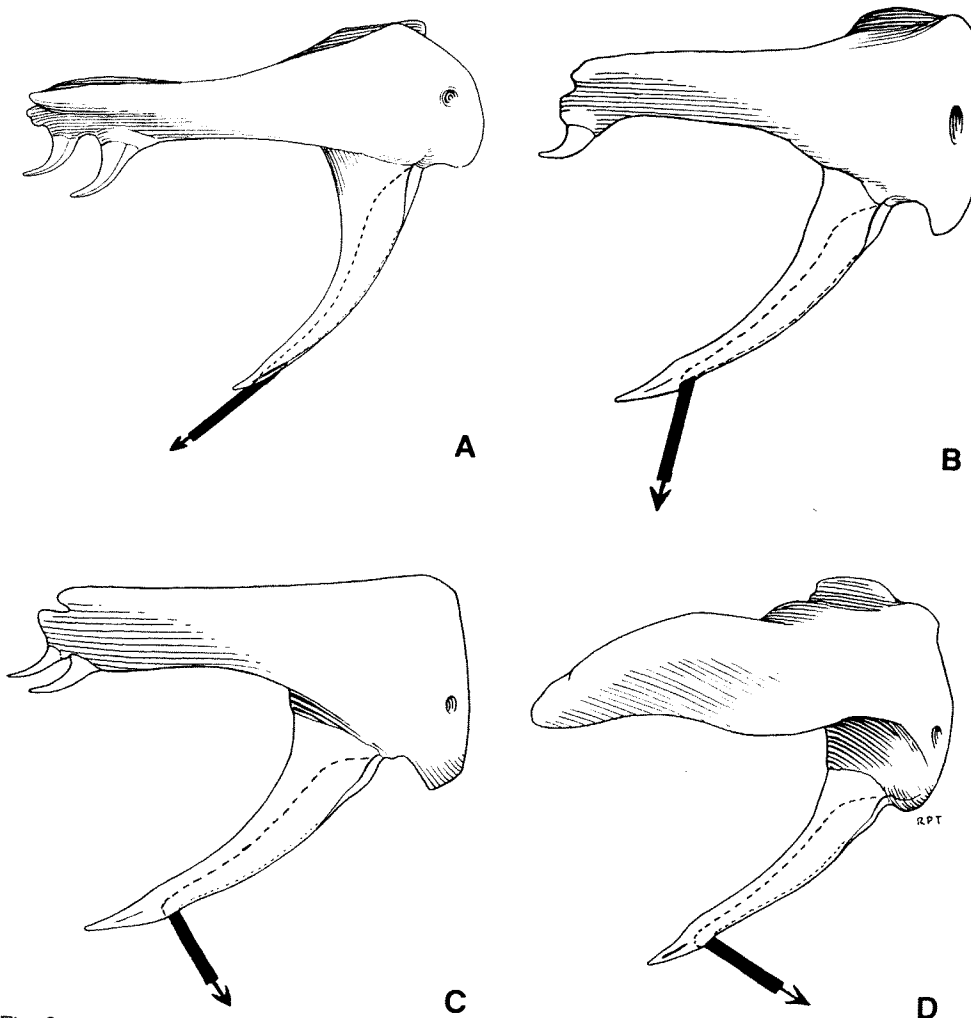


Fig. 8-12. Lateral views of some representative types of cobra fangs. (*A*) *Naja melanoleuca*, a non-spitter. (*B*) *N. naja*, a Chinese cobra partly adapted for spitting. (*C*) *N. nigricollis*, the spitting cobra. (*D*) *Hemachatus haemachatus*, the ringhals. Bogert considers the fangs of the last species to be the most perfectly adapted for spitting.[19]

of 12 feet. Loveridge noted a figure of approximately 6 feet,[17] while Noyes stated that he has "never known the ringhals to spit at an object outside the range of his spray,"[22] which answers the question nicely. I once had a very large *N. nigricollis* that ejected venom 157 cm. in the laboratory. Warrell and colleagues reported a figure of 5 meters.[23] Joan Root, as reported to Charles Bogert in 1978, believes that the snake's aim is pretty accurate up to a distance of about 8 feet.

This modification in the fangs of the spitting cobras appears to have evolved gradually. Bogert noted the following:[19]

> Comparisons of the fangs of spitters and non-spitters indicate that those of the former evolved from fangs not unlike those of the latter, by partial closure of the lower end of the discharge orifice and the loss of the groove below the orifice that extends onto the point of the fang in non-spitters. Intermediate stages are represented only in *Naja naja,* while available specimens of *Hemachatus* and *N. nigricollis* are uniformly well adapted to expel venom forward from the fang. The latter two species are likewise more inclined to use their venom for defense, whereas the spitting behavior cannot be evoked in many specimens of *Naja naja.*
>
> Therefore, it appears to be possible that, following an original fortuitous modification, the fangs of spitting cobras have become perfected as a result of small cumulative orthogenetic modifications probably directed by natural selection. . . . On zoogeographical and anatomical grounds it can be stated that adaptive fang characters have evolved separately in three* distinct but related species. Similar, but in no case identical, fang adaptations are not indicative of close relationship, however, but have resulted from parallel evolution in originally diverging strains of a common ancestral stock.

As Bogert has noted, any cobra might be capable of using its venom in a defensive manner, if it could evolve the necessary behavioral and fang adaptions.[19] In the crotalids and viperids, the fang structure is unsuited for spitting, and the venom does not appear to be absorbed through the conjunctiva, at least under laboratory conditions. The relationship between fang structure and conjunctival venom absorption in the spitting cobras, and between fang structure and relative lack of venom absorption in the viperids and crotalids seems significant.

The question of what or where these cobras aim their venom has not been answered. Most writers on spitting cobras discuss the "accuracy" of the spit, but this accuracy is not defined. That they can spit in the eyes of man, there is no question, but whether they aim at the eyes, face, or head, or at movement, brightness, or dullness, or at some "end" of a mammal has not been determined. Among other things, it is not known how often they miss. It has been suggested that spitting cobras are able to differentiate body surface temperatures and thus distinguish the temperature of the face, free of hair, from that of the surrounding areas or covered part. Other writers have sug-

*Now there are four species with the recognition of *Naja mossambica.*

gested that they spit at movement or at the closest part of an animal's movement, which, in most cases in which they are challenged, would be the head. Still others suggest that the snake aims at the eyes, because so many animals center their attention on other animal's eyes.* It must be admitted, however, that there is no experimental evidence to explain the aim of the venom.

SYMPTOMS AND SIGNS OF POISONING

When the venom is sprayed into the eyes of a human there is almost instantaneous pain. In rabbits in which the venom has been immediately washed from the eye, we have observed no local tissue changes. However, in both humans and experimental animals, if there is a delay in irrigating the eye, some conjunctivitis develops. The severity is related, in part, to the length of exposure to the venom. If irrigation of the eye is delayed, or if the irrigation is ineffective, other eye changes may occur in addition to conjunctivitis. In the several patients I have seen, there was mild conjunctivitis, edema of the eyelid, and pain, which in one patient radiated from the involved right eye over that side of the face. Ocular tension was normal, and there was no blepharitis or evidence of corneal ulcers. The anterior chamber, pupil, iris, and lens were normal. The patient complained of a dull headache for 3 days.

Warrell and Ormerod reported on nine patients having *Naja nigricollis* ophthalmia: five developed conjunctivitis, four had corneal ulceration, one developed anterior uveitis, and two were permanently blinded.[24] Other descriptions of spitting cobra ophthalmia have been provided by Pergola[25] and Sarnelli.[26]

Bites by *Naja nigricollis* give rise to pain, which is often immediate, bleeding from the wound site, localized swelling, which may spread to involve the entire limb within several hours, and some ecchymosis (Fig. 8-13). Blebs and blood-filled blisters may form. Necrosis has been reported,[23] although in our two patients this did not develop. Leukocytosis, a fall in hematocrit, and thrombocytopenia may occur. Clot retraction time is often prolonged. Fibrinogen levels were normal in my two patients. Vomiting has been reported, although in my limited experience this was not observed; mild nausea was present, however. Drowsiness was present in both of our patients.

There was a definite muscle weakness in the affected extremity, with a slight decrease in deep reflexes in one patient. The same patient complained of a sensation of "pins and needles" over the injured part. He also had some decrease in muscle strength in that part. It is certain that both of our patients were lethargic and considerably more drowsy than patients I have observed following rattlesnake bites. Deep reflexes were decreased over the involved extremity in one patient, and he developed paresthesia and muscle weakness over the affected arm and forearm. Since our cases were seen within 1 hour

*Bogert, C. M.: Personal communication, 1977.

Fig. 8-13. Local tissue effects 10 hours after a bite by *Naja nigricollis*.

following the bite and antivenin was given promptly, perhaps further significant neurological findings failed to develop. However, Warrell and Ormerod have had far more experience with treatment of bites by this species than I, and other than drowsiness, their examinations have failed to uncover any significant neurological deficits.[23]

TREATMENT OF POISONING

Immediate irrigation of the involved eye (or eyes) with water is imperative. As soon as possible, the eye should be irrigated with normal saline solution, and a 1.5-per-cent Neo-Cortef ointment, instilled 3 times per day for several days. In most cases, this treatment will suffice. However, the eye should be examined daily and any changes treated, as indicated. Instillation of specific antivenin has not been shown to be of value, although its use is advocated by some physicians.

For the treatment of envenomation by this species, immobilization of the affected part in a functional position, administration of intravenous fluids,

preferably albumin, and complete bed rest are essential. Antivenin should be given whenever swelling extends beyond the injured area. As experiences reported in the literature indicate, too little antivenin has usually been given, and often given too late, to people bitten by this snake. In both of the cases treated by the author, a good response was obtained with the Institut Pasteur Anti-Bitis-Echis-Naja antivenin. I employed 9 vials in one patient and 8 vials in the other. There was no immediate or delayed serum reaction in either of these patients.

Cobra
(Naja sp.)

The cobras are remarkably different in their size, behavior, color and markings, fang types, and their venoms. Six species are generally recognized and all are African, except the Indian or Asiatic cobra, *Naja naja* (Fig. 8-14). They range in size from 4 to 9 feet (1.2–2.7 meters). The king cobra *(Ophiophagus hannah)* may measure up to 14 feet (4.3 meters). When cornered, cobras rear up and spread their hoods, and they may strike if molested. However, in striking, the mouth is often closed. In biting, they tend to hold on and may sometimes chew savagely. Some cobras may spit or spray their venom (see pp. 353–361).

The cobras are easily distinguished from other snakes, and it is not likely that an error in identification will be made in U.S. collections. Fortunately, approximately 50 per cent of cobra bites in humans do not end in envenomation, so definitive treatment is often unnecessary.

SYMPTOMS AND SIGNS OF ENVENOMATION

The symptoms and signs of cobra venom poisoning appear to vary considerably. In the patients seen by us, they have been rather consistent, but in reviewing the literature, there appears to be a farrago of descriptions. Part of the difficulty can be attributed to the differences in the venoms of the different species and subspecies, part to the possible misidentification of the snake, part to poor observations, and certainly part to the unsatisfactory method of dividing snakebites in Asia and Africa into "neurotoxic," "cardiotoxic," and "hemotoxic" cases.

In most cobra bites, symptoms and signs will appear between 1 and 4 hours. Bilateral ptosis is the earliest and commonest manifestation. Respiratory difficulties and failure may follow in some patients, with ophthalmoplegia, glossopharyngeal palsy with increased saliva, paresis of the neck and limbs, and the development of flaccid paralysis and coma. Convulsions, shock, and cardiac arrest occur in some patients.

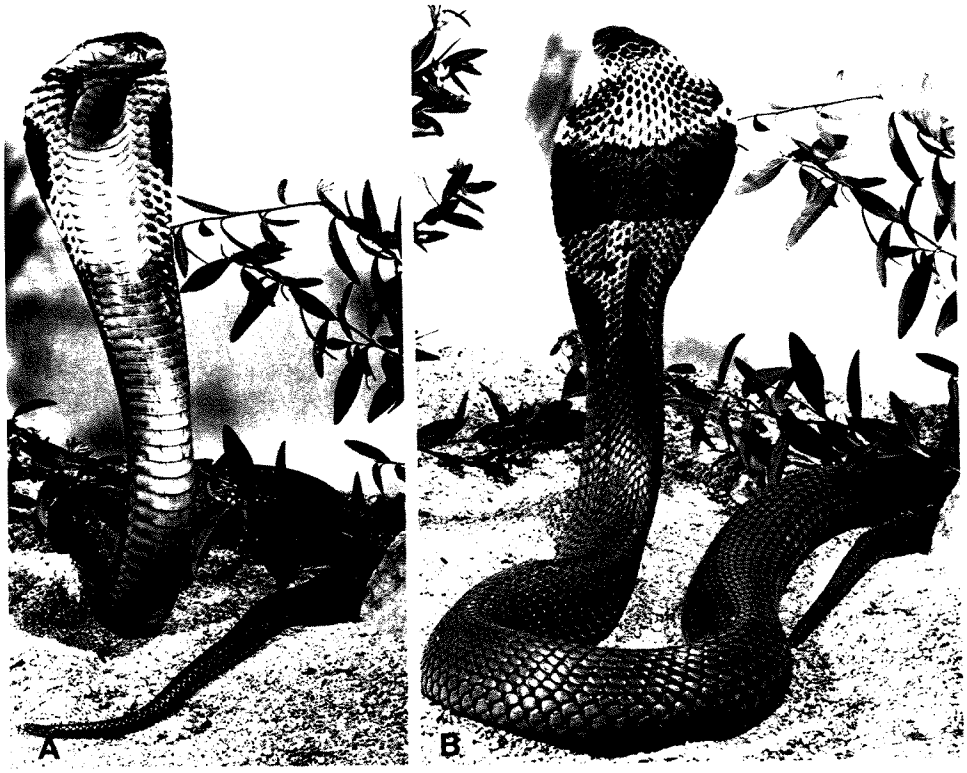


Fig. 8-14. (*A,B*) The Indian cobra, *Naja naja naja*

In our patients, there has been no local tissue damage (Fig. 8-15). One of the first symptoms was a feeling of intoxication or drowsiness, and there was usually some local pain. Ptosis developed, but in our opinion, the prompt use of antivenin, ventilation, and other supportive measures blocked the development of additional significant manifestations.

In Malaya, Reid noted that local swelling and necrosis developed in 35 per cent of envenomated patients and overshadowed the neurotoxicity of the venom (Fig. 8-16). The subspecies implicated in these bites were *Naja naja kaouthia* and *N. n. leucodera.*[27] The development of severe local tissue changes with few or no significant neurological manifestations following bites by some cobras was also noted by earlier workers, including Moore,[28] Bull,[29] and Hennessy.[30]

Recently, I treated a patient bitten by *N. n. leucodera.* The patient was first seen 3 days following the bite, and at this time there was still some residual swelling, with one large, blood-filled blister on the left ring finger, and mild ecchymosis over the dorsum of the hand. Debridement revealed some necrosis of the subcutaneous tissues (Fig. 8-17). The patient was treated with

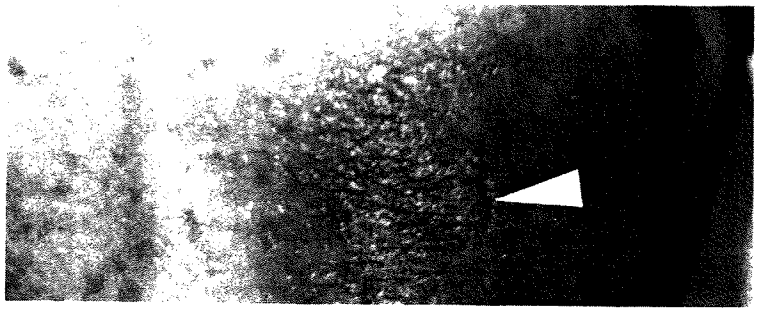


Fig. 8-15. Wound area 2 days following bite by *Naja naja naja*. The victim had joined two fang marks with an incision. Note some residual ecchymosis.

Fig. 8-16. Local tissue effects several days after a bite by a cobra.[27]

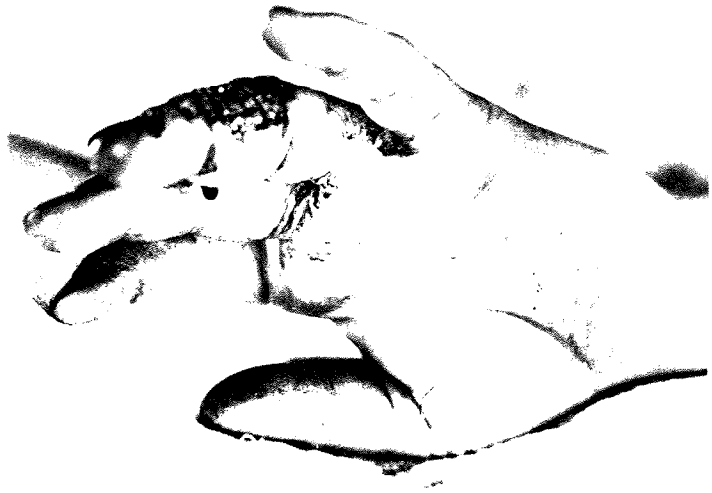


Fig. 8-17. *Naja naja leucodera* bite. Localized, superficial tissue damage is apparent around area of bite.

Burow's soaks, local oxygen, and physical therapy. He recovered completely, except for the complaint of drowsiness, weakness, and fatigue of 6 days duration, for which I have no explanation.

These various experiences again indicate the care that must be taken by both clinicians and researchers in properly identifying the offending snake or the experimental venom. It may not be sufficient to merely note the reptile as *Naja naja.*

TREATMENT OF ENVENOMATION

Treatment of *Naja naja naja* bites consists of antivenin, intravenous solutions, and ventilation, sometimes with tracheostomy. The author has used a minimum of five vials of the Haffkine Institute antivenin in one case and a maximum of 17 vials in a serious case. Banerjee and colleagues have advised the use of 0.5 mg. neostigmine, intravenously, immediately following the appearance of the first neurological signs; then, 0.5 mg. every 30 minutes, for 5½ hours; and finally, the same dose every 2 to 12 hours, consistent with the rate of recovery, along with the antivenin.[31] The author has had no experience with this drug, but it should be tried in view of Banerjee's observations.* It has not been found of value in sea-snake bites, nor in a patient bitten by *Bungarus candidus.* Apparently, it has not been used in the treatment of Malayan cobra venom poisoning.†

The use of steroids, antihistamines, heparin, and other drugs has not proved of value in the therapy of cobra venom poisoning, although they have been widely employed and, sometimes, heralded. When there is a respiratory deficit, the patient should be placed in a semi-upright position. Intubation and ventilation should be initiated, and any excessive salivary secretions suctioned off. A tracheostomy may be necessary in some cases. Electrolyte balance should be maintained and the necessary supportive measures instituted.

The treatment of bites by those cobras with venoms that produce greater local tissue reactions than neurological disturbances is controversial. It is frequently stated that cobra antivenins do not protect against the local tissue-necrotizing properties of certain cobras. This is true, in part. In experiments on rats, we found that most cobra antivenins afforded either no or only minimal protection against the local tissue activity of certain cobra venoms. However, the antivenin produced in Indonesia (Bio Farma) does appear to neutralize, in part, the fraction or fractions, responsible for the tissue destruction, at least in rats. This antivenin is specific for *Naja naja sputatrix* but it also seems effective, experimentally, against *N. n. leucodera* and *N. n. kaouthia* venoms.

*Banerjee, R. N.: Personal communications, 1978.
†Reid, H. A.: Personal communication, 1978.

The application of Reid's statement (that the Haffkine and Queen Saovabha antivenins do not "prevent or ameliorate local necrosis[27]) is unwarranted as a sweeping generalization of the efficacy of all antivenins on the local tissue reaction. This generalization has occurred in the literature in the United States, and elsewhere. The choice of the proper cobra antivenin must be carefully considered. The therapeutic deficiencies frequently attributed to the antivenins can be more easily explained on the basis of an improper choice of an antisera, or the use of an insufficient quantity, in most instances. Whenever there is a question about the use of an antivenin, the physician should seek consultation from the *Antivenin Index* (405-271-5454) in Oklahoma City. The efficacy of the various antivenins prepared for bites by exotic species has been the object of study by Dr. Sherman A. Minton of the University of Indiana School of Medicine. Professor H. A. Reid of the Liverpool School of Tropical Medicine is also an advisor in this capacity.

Fer-De-Lance

(*Bothrops lanceolatus, B. atrox,* and *B. asper)*

The name Fer-de-lance is given to many species of *Bothrops* by collectors and amateur herpetologists in the United States. The true Fer-de-lance is *B. lanceolatus,* but it is not uncommon to see the name used for *B. asper, B. atrox,* and even *B. caribbaeus. B. lanceolatus* is a large, somewhat hyperactive snake of 4 to 6 feet (1.2–1.8 meters) in length, with occasional individuals reaching 7 feet (2.1 meters). It is limited to the Island of Martinique and is the only venomous snake found there. It has the typical lance head of the *Bothrops* species. The head is marked with dark, truncated, lateral blotches and a sharply defined, dark brown, postorbital band that extends down to the back of the mouth. The body is brown, gray, or olive, with an obscure series of 22 to 27 hour-glass-shaped blotches along the back. This snake has very long fangs.

Bothrops atrox is slightly larger and may attain a length of over 8 feet (2.4 meters; Fig. 8-18). It is olive green, gray, or brown in color and is marked with a pattern of 20 to 30 blackish-edged triangles, having a lighter center, with apices that meet near the vertebral line. It is a very common snake throughout its range from Colombia south to Peru, Brazil, and the northeast part of Argentina. It is probably the most common biting venomous snake throughout most of its range.

SYMPTOMS AND SIGNS OF ENVENOMATION

The symptoms and signs of bites by the various *Bothrops* species are somewhat similar, but there are some important differences. Generally, there is some pain, and it may be severe immediately following the bite. Erythem-

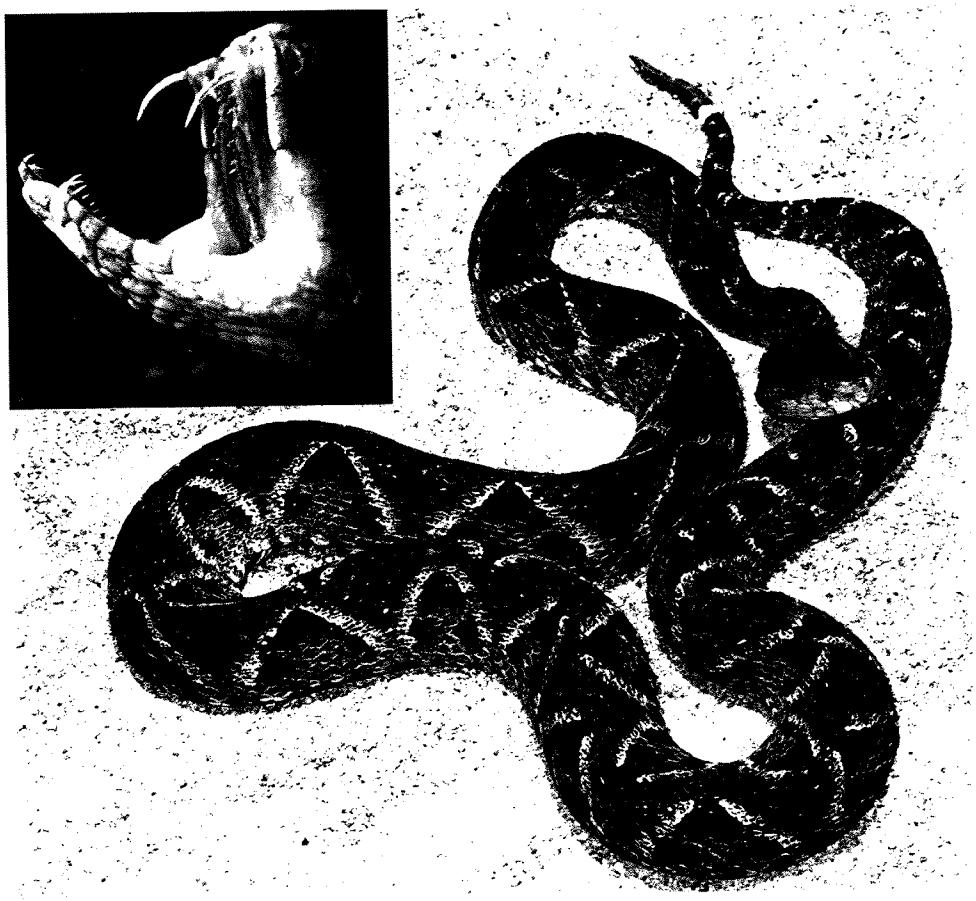


Fig. 8-18. Bothrops atrox, Barba Amarilla (Fer-de-lance). The fangs are shown in the insert.

atous edema and ecchymosis develop (Figs. 8-19 and 8-20). The ecchymosis may often be seen along blood vessel paths in the affected part. Petechiae may develop in various parts of the body, and clusters of reddish spots may appear over the involved extremity. Neither edema nor bleb formation are as marked as in rattlesnake bites, but necrosis can occur. The venom has a direct effect on blood cells with red blood cell lysis and thrombocytopenia occurring in the moderately or severely envenomated patients. Rosenfeld and colleagues stated that when incoagulable blood is found 1 hour following envenomation, more than 0.1 mg. per kg. of venom may have been injected.[32] Dizziness, nausea, vomiting, hematemesis, hematuria, melena, epitaxis, and gingival bleeding are common findings.

Fig. 8-19. Fer-de-lance bite of the hand shows edema and ecchymosis.

Fig. 8-20. Bothrops atrox bite shows edema and erythema.

TREATMENT OF ENVENOMATION

Treatment consists of intravenous antivenin, intravenous fluids, immobilization of the affected part in a functional position, rest, and a program similar to that suggested for rattlesnake venom poisoning. Our antivenin experiences have been with the Wyeth (Crotalidae) Antivenin and with Sôro Antibotropico of the Instituto Butantan. In the case of the former, we administered 7 vials, and in the latter, 5 to 7 vials. Delayed serum reactions occurred in all patients.

South American Rattlesnake
(Crotalus durissus terrificus)

The South American rattlesnake must be considered the most dangerous of the rattlesnakes. It is a large, stout, bad-tempered reptile, 4 to 5 feet (1.2–1.5 meters) in length, and sometimes reaches 6 feet (1.8 meters; Fig. 8-21). Its venom glands contain 20 to 100 mg. of dried venom, with an intravenous LD_{50}, in mice, of 0.30 mg. per kg. Thus, the patient bitten by this crotalid must be treated quickly and diligently. The dorsum is brown or olive and marked with large, darker, light-edged rhombic blotches or diamonds. The tail is often unicolor brown or black.

SYMPTOMS AND SIGNS OF ENVENOMATION

Pain, often stinging in character, is a consistent complaint following the bite of this snake. The pain may be followed by a feeling of numbness over the affected part. Edema rarely develops, and ecchymosis, if it occurs, is limited to the area of the bite. Bleb formation does not occur (Fig. 8-22). Visual disturbances develop within 1 hour of the bite, and ophthalmoplegia and blepharoplegia develop soon after, in some cases. Pupillary reflexes are usually intact. Rosenfeld noted the presence of "the neurotoxic facies," which is diagnostic of *C. d. terrificus* bites (Fig. 8-23).[33] Muscle pain and weakness may develop.

Paresis may be most notable in the muscles of the back of the neck. In the two cases seen by the author, fine muscle fasciculations were observed in the neck and face, although they were most notable over the tongue. In severe poisoning, there may be vomiting, decreased deep reflexes, prostration, and coma. Methemoglobinuria may occur within 6 hours of the bite and is often followed by anuria in the more severe envenomations. Pulse and blood pressure may be normal until late in the course of the poisoning. The hemogram is usually within normal limits, although late in the poisoning, the blood may become incoagulable. Death has been attributed to the "nephrotic syndrome."[34]

Fig. 8-21. South American rattlesnake, *Crotalus durissus terrificus.*

TREATMENT OF ENVENOMATION

The immediate use of adequate amounts of antivenin has been advised by most clinicians experienced with these poisonings. According to Rosenfeld,* the prompt use of antivenin will prevent the neurological manifestations from developing. However, once symptoms appear, the use of antivenin will only slowly relieve the deficit. The author has used both the Instituto Butantan, Anticrotalico product and the Wyeth Antivenin (Crotalidae) Polyvalent. With the former, 100 ml. (100 mg.) was used, while with the latter, 10 vials were employed. The antivenin should be given intravenously in a drip of 5-percent glucose in water. The appropriate anti-tetanus agent should be given, and the patient hospitalized. Steroids, ice, prostigmine, and exsanguino-transfusions are reported to be of no value.[34]

*G. Rosenfeld: Personal communication, 1969.

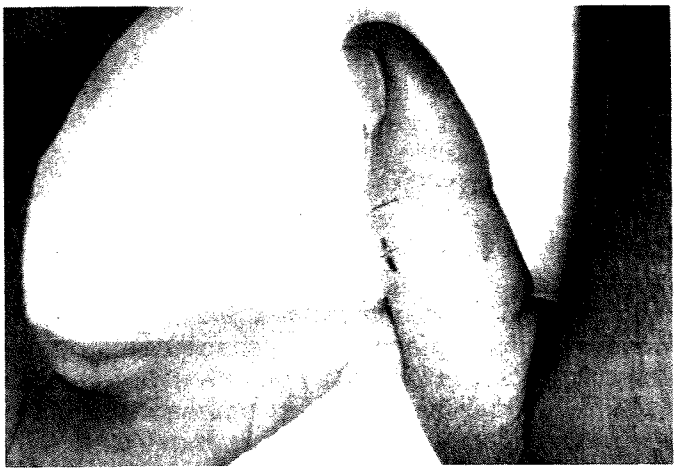


Fig. 8-22. Bite by South American rattlesnake shows fang marks and minimal edema.

Fig. 8-23. Facies seen after serious envenomation by a South American rattlesnake.[33]

Tiger Snake
(Notechis scutatus)

The adult tiger snake is 4 to 5 feet (1.2–1.5 meters) in length, although specimens up to 8 feet (2.4 meters) have been reported. This extremely dangerous reptile varies in its ground color from yellowish, greenish-gray, olive, or brown, to almost black, with creamish-yellow or gray transverse bands. Most commonly, its ground color is brownish-gray. In some specimens the bands are indistinct, and the snake appears to be one color (Fig. 8-24). The head is somewhat flattened dorsoventrally and is only slightly distinct from the neck. This snake is chiefly nocturnal and is commonly found in grass or in low shrubbery, particularly near marshy areas.[35,36]

SYMPTOMS AND SIGNS OF ENVENOMATION

Although the bite area may show two teeth or fang marks, it is not uncommon to find multiple tooth punctures. These may be difficult to locate, particularly since there is minimal local tissue reaction. There may be bleeding from the wound site, and this can persist. In most cases there is some delay in the appearance of significant clinical manifestations. In one case seen by the author, the first signs of envenomation were dizziness, headache, and abdominal pain, approximately 30 minutes following the bite. Until that time the patient did not believe that he had been envenomated. Examination revealed slightly enlarged and tender axillary lymph nodes on the affected side, mild weakness of the arm (at 90 minutes), and the complaint of some feeling of numbness over that extremity. Reflexes were intact at that time. The patient complained of weakness, drowsiness, and headache.

Shortly thereafter, the patient developed pain in the abdomen, in the large muscle masses of the back and shoulders, and in the chest on inspiration.

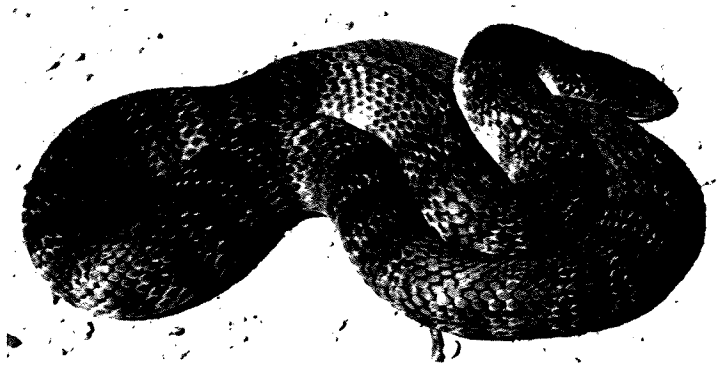


Fig. 8-24. Tiger snake, *Notechis scutatus.*

There was moderate weakness of the muscles of the upper extremities and shoulders. Since antivenin was given at this time, no further manifestations developed except for hematuria, which persisted for 12 hours. The patient had an increased blood-clotting time and decreased blood calcium, but these deficits disappeared within 48 hours.

In a second case, the patient complained of headache and some muscular weakness within 15 minutes of the bite. At 30 minutes, he noted slurring of speech and onset of dull abdominal pain. At 45 minutes, he had some difficulty in breathing, some blurring of vision, difficulties in focusing, slight ptosis, and gradually developing paresis. These findings worsened over the next 8 hours. The abdominal pain became severe, and both the urine and stools contained bright red blood. Skeletal muscle paralysis developed.

At this point, approximately 30 hours following the bite, a tracheostomy was performed and artificial ventilation initiated (Fig. 8-25). Blood electrolytes and the blood clotting screen were studied, and the patient was given three pints of fresh whole blood. Electrolyte imbalances were corrected. Serum enzyme studies were normal and there was no myoglobinuria. An artificial pacemaker was placed because of cardiac arrhythmia. The patient had a slow but uneventful recovery, although blood electrolyte and clotting studies

Fig. 8-25. Patient in respiratory paralysis showing paresis and paralysis of facial muscles. The patient is responding to being asked to "wrinkle" his forehead.

varied considerably from day to day and necessitated constant attention. The patient had a deficit of the olfactory nerve for many months following the bite.

The symptoms and signs of bites by elapids have been reviewed by Campbell[37] and Sutherland.[38] Unfortunately, their descriptions are not separated on the basis of the specificity of the snake, but this is not always possible. For Australian elapid bites, in general, they noted headache, nausea, vomiting, abdominal pain, hypotension, loss of consciousness, ptosis, blurred or double vision, facial and pharyngeal paralysis, generalized muscle weakness and paralysis, lymphadenitis, hemorrhage, hematuria, hemoptysis, hematemesis, and respiratory distress. Hood and Johnson noted myoglobinuria in a patient following a tiger snake bite;[39] and, Harris and colleagues described a presynaptically active fraction of the venom, called a neurotoxin, which also destroys skeletal muscle.[40] Approximately 45 per cent of all tiger snake bites were fatal before the advent of antivenin.[41]

TREATMENT OF ENVENOMATION

If any significant symptoms or signs of envenomation develop, intravenous antivenin should be given. The antivenin of choice is the Commonwealth Serum Laboratory, tiger snake antivenin. With minimal poisoning, 6,000 units (2 vials) should be administered; moderate envenomations may require 12,000 to 15,000 units, while severe envenomations may necessitate 30,000 units. In the severely envenomated patient seen at our Medical Center, I used 21,000 units of antivenin. It is not known how long after envenomation this antivenin can be given and still be effective, but I would advise its use up until at least 48 hours after the bite.

The rapid onset of respiratory paralysis in one of our cases emphasizes the stress Campbell places on tracheostomy. Of his 73 patients bitten by elapids, tracheostomy was done in 32 cases. The approximate average time from bite to institution of the procedure was 21 hours, with a range of 3 to 96 hours.[42] In both of our cases, intubation and ventilation were initiated, and excessive salivary secretions were suctioned. Since no vomiting occurred, a Levine tube was not passed. In one patient we performed a tracheostomy.

The blood changes in humans envenomated by the tiger snake are somewhat confusing. The reader should consult Sutherland's paper for a discussion of this problem.[43] Until these changes are better understood, it is best for the physician to follow the bleeding, clotting, and prothrombin times, and the hematocrit and fibrinogen levels, closely. Sutherland advises serum enzymes, electrolyte, and electrocardiographic studies.[44]

Intravenous fluids, preferably albumin, are essential. Shock drugs may need to be administered. Morphine should be avoided, and steroids should be limited to the treatment of serum reactions.[38]

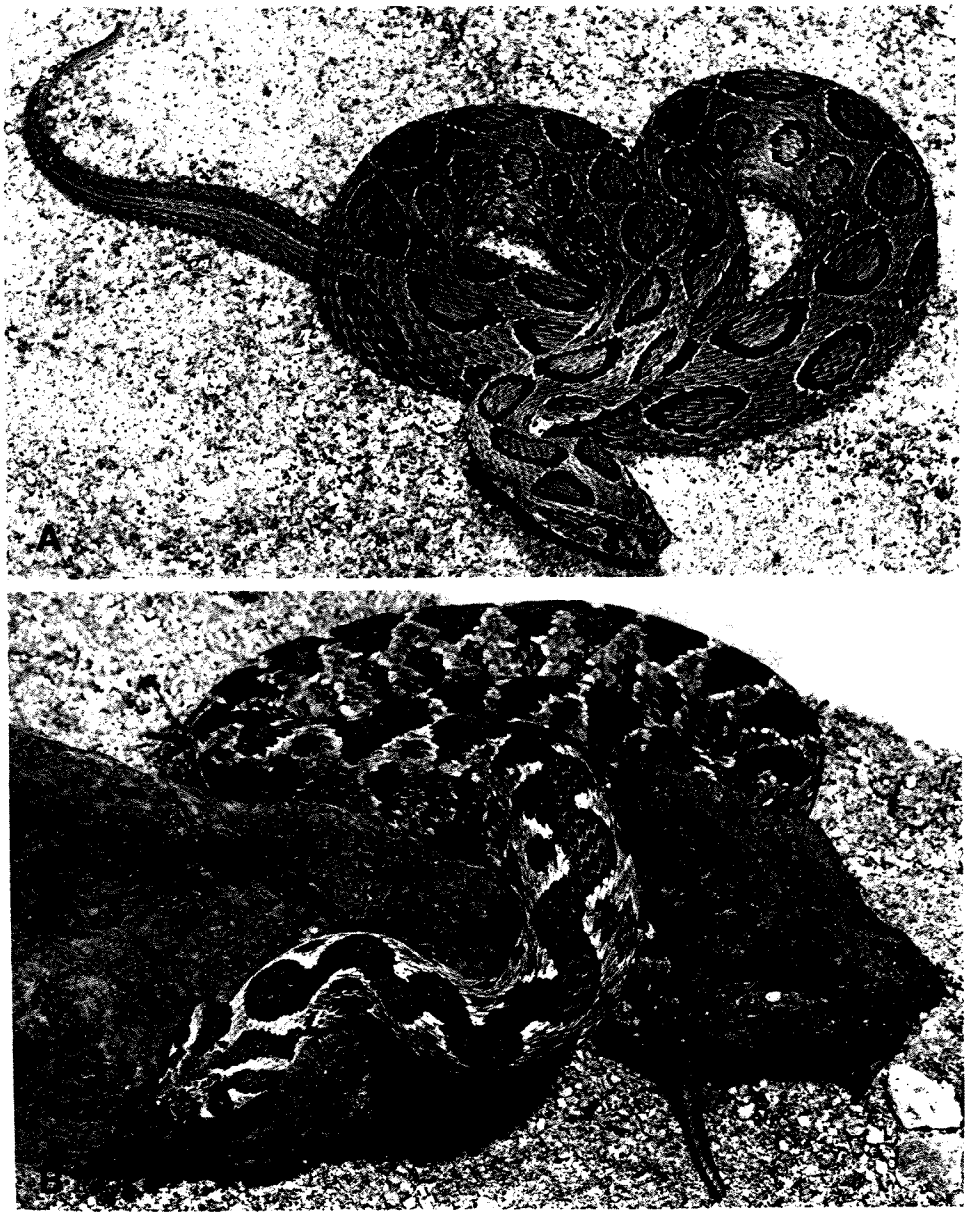


Fig. 8-26. (*A*) Russell's viper, *Vipera russelli.* (*B*) Palestine viper, *Vipera xanthina palaestinae.*

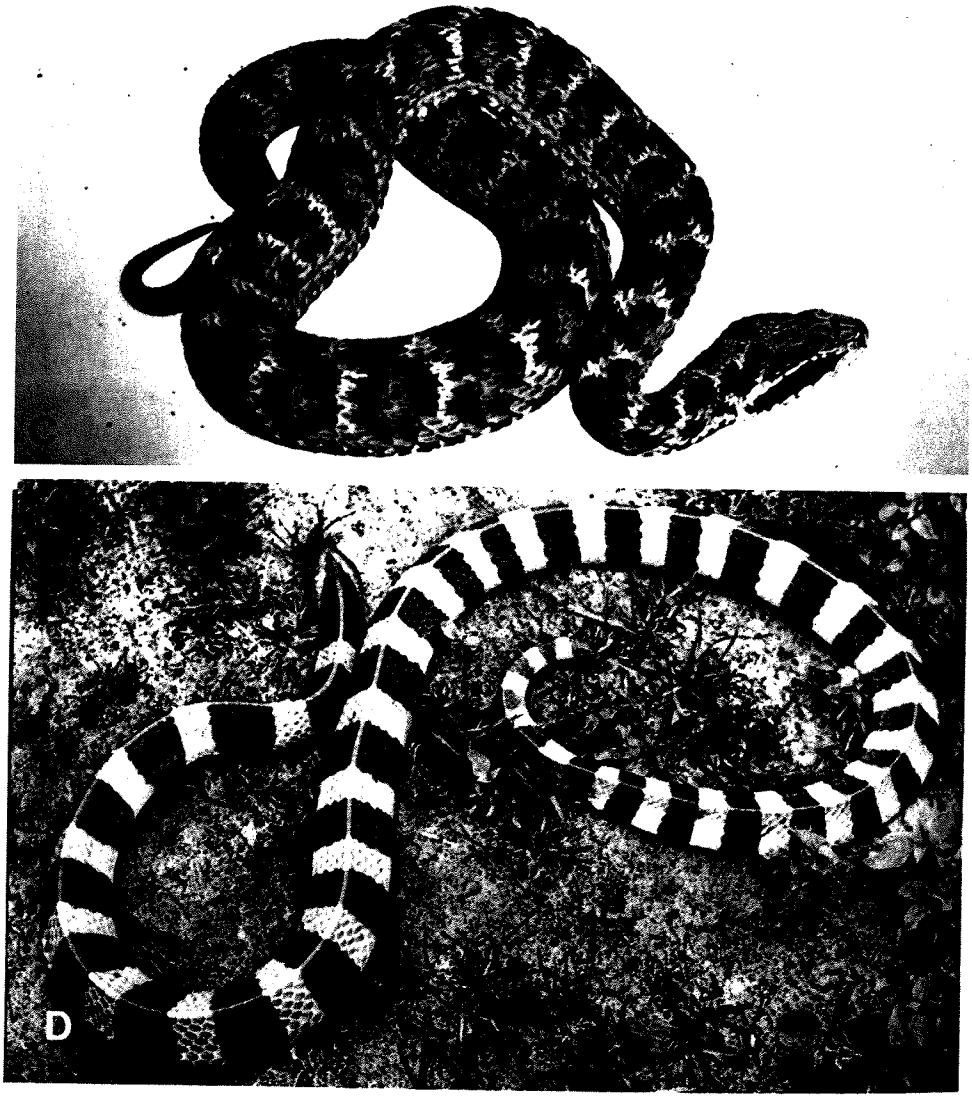


Fig. 8-26 (continued). (C) Mamushi, *Agkistrodon halys.* (D) Banded krait, *Bungarus fasciatus.*

Antivenins

A listing of available antivenins for the treatment of envenomations by exotic snakes is given below in Table 8-2. This listing is updated from that authored by Russell and Lauritzen,[45] to which the reader is directed for a more detailed discussion of antivenins. *(Text continued on p. 395)*

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
North America					
Wyeth Laboratories Box 8299, Philadelphia, Pennsylvania, U.S.A.	*Crotalus durissus terrificus* *Bothrops atrox* *Crotalus adamanteus* *Crotalus atrox*	Antivenin (Crotalidae) Polyvalent	South American rattlesnake Barba Amarilla Eastern diamondback rattlesnake Western diamondback rattlesnake	*Crotalus* sp. *Sistrurus* sp. *Agkistrodon* sp. (Old & New World) *Bothrops* sp. *Lachesis* sp. *Trimeresurus* sp.	Precipitated with ammonium sulphate, and lyophilized
	Micrurus fulvius fulvius	Antivenin (*Micrurus fulvius*)	Eastern coral snake	*Micrurus fulvius tenere*	
Laboratorios "MYN", S.A. Av. Coyoacan 1707 Mexico City 12, D.F., Mexico	*Bothrops atrox asper*	Monovalent Bothrops	Terciopelo		Enzyme digested, precipitated with ammonium sulphate, and lyophilized
	Crotalus atrox *Crotalus d. terrificus* *Crotalus tigris*	Polyvalent Crotalus	Western diamondback rattlesnake South American rattlesnake Tiger rattlesnake	All Mexican crotalids	
	Bothrops atrox asper *Crotalus d. terrificus* *Crotalus tigris* *Crotalus atrox*	Polyvalent Mexico	Terciopelo South American rattlesnake Tiger rattlesnake Western diamondback rattlesnake	All Mexican crotalids	

(continues)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
North America					
Instituto Nacional de Higiene, Csda. M. Escobedo No. 20, Mexico City, D.F., Mexico	*Bothrops atrox asper*	Anti-Bothrops	Terciopelo		Ammonium sulphate precipitation. Supplied in liquid form.
	Crotalus basiliscus	Anti-Crotalus	Mexican rattlesnake		
	Crotalus d. terrificus	Anti-Crotalus	South American rattlesnake		
	Bothrops atrox asper	Polyvalent	Terciopelo		
	Crotalus b. basiliscus	Polyvalent	Mexican rattlesnake		
	Crotalus d. terrificus	Polyvalent	South American rattlesnake		
Central and South America					
University de Costa Rica Ciudad Universitaria Rodrigo Facio San Jose, Costa Rica	*Lachesis muta stenophyrs*	Anti-Laquesico	Bushmaster	*Lachesis muta muta* *Lachesis muta noctiyaga*	Precipitated with ammonia sulphate Freeze dried or liquid
	Bothrops atrox asper	Polyvalent	Terciopelo	*Lachesis muta muta*	
	Crotalus durissus durissus	Polyvalent	Central American Rattlesnake	*Lachesis muta noctiyaga*	
	Lachesis muta stenophyrs	Polyvalent	Bushmaster	*Agkistrodon bilineatus* *Bothrops nummifer* *Bothrops picadoi* *Bothrops nasutus* *Bothrops ophryomegas* *Bothrops godmanni* *Bothrops lateralis* *Bothrops schlegelii* *Bothrops nigroviridis*	

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Central and South America					
	Micrurus nigrocinctus nigrocinctus *Micrurus nigrocinctus mosquitensis*	Anti-Coral (Central America)		*Micrurus carinicaudus* *Micrurus dumerilii* *Micrurus fulvius fulvius*	
	Micrurus nigrocinctus *Micrurus mipartitus* *Micrurus frontalis*	Anti-Coral Polyvalent		*Micrurus fulvius fulvius* *Micrurus alleni* *Micrurus carinicaudus* *Micrurus spixii* *Micrurus lemniscatus* *Micrurus corallinus*	
Instituto Nacional de Salud Ave. Eldorado con Carrera, Zona G, Bogota, D.E., Colombia	*Bothrops atrox* *Crotalus d. terrificus*	Antiophidico Polivalente	Barba Amarilla South American rattlesnake	*Bothrops* species *Crotalus* species	Globulin precipitated with ammonium sulphate
Laboratorio Behrens Ave. Principal de Chapellin, Apartado 62, Caracas, 101 Venezuela	*Crotalus d. terrificus* *Bothrops atrox* *Bothrops venezuelae*		South American rattlesnake or cascabel Barba Amarilla Tigra-mariposa	*Crotalus vegrandis* *Bothrops colombiensis*	Foreign-protein-reduced

(continues)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Central and South America					
	Bothrops atrox *Bothrops venezuelae* *Crotalus d. terrificus*		Barba Amarilla Tigra-mariposa South American rattlesnake or cascabel	*Bothrops colombiensis* *Bothrops bilineata* *Bothrops lansbergii* *Bothrops lichenosus* *Bothrops medusa* *Bothrops neglectus* *Bothrops schlegelii* *Crotalus vegrandis*	
Instituto Nacional de Microbiologia Avdo. Velez Sarsfield 563, Buenos Aires, Argentina	*Crotalus d. terrificus*		South American rattlesnake or cascabel	Purified by enzymatic and differential thermocoagulation techniques (No recent confirmation)	
	Bothrops alternatus *Bothrops neuwiedii*	Bothrops Bi-Valent	Yarara or de la Cruz Wied's lance-head, Yarara Chica or painted jararaca		
	Bothrops alternatus *Bothrops jararaca* *Bothrops jararacussu* *Bothrops neuwiedii* *Crotalus d. terrificus*	Tropical Polyvalent	Yarara or de la Cruz Jararaca Yarara Wied's lance-head South American rattlesnake or cascabel		

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Central and South America					
	Bothrops alternatus *Bothrops neuwiedii* *Crotalus d. terrificus*	Tropical Tri-Valent	Yarara or de la Cruz Wied's lance-head South American rattlesnake		
Instituto Butantan Ciaxa Postal 65, São Paulo, Brazil	*Crotalus d. terrificus*	Anticrotalic	South American rattlesnake or *cascabel*	It can be expected that the antivenins of this institute neutralize other crotalid venoms, even though the producers note in a personal letter that the scarcity of data preclude any specific claims.	Pepsin-digested, and ammonium sulfate precipitation
	Lachesis muta	Antilaquetico	Bushmaster or Surucucu		
	Bothrops jararaca *Bothrops moojeni* *Bothrops cotiara* *Bothrops alternatus* *Bothrops jararacussu* *Bothrops neuwiedi*	Antibothropico	Jararaca Moojen's pit viper Cotiara Urutu Jararacussu Wied's lance-head or painted jararaca		
	Crotalus d. terrificus		South American rattlesnake		
	Bothrops jararaca *Bothrops moojeni* *Bothrops cotiara* *Bothrops alternatus* *Bothrops jararacussu* *Bothrops neuwiedi*	Antiophidico Polyvalent	Jararaca Moojen's pit viper Cotiara Urutu Jararacussu Wied's lance-head or painted jararaca		

(continues)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Central and South America					
	Lachesis muta	Antibothropico-lachetico	Bushmaster		
	Bothrops alternatus		Urutu		
	Bothrops jararacussu		Jararacussu		
	Bothrops jararaca		Jararaca		
	Bothrops moojeni		Moojen's pit viper		
	Bothrops cotiara		Cotiara		
	Bothrops neuwiedi		Wied's lance-head or painted jararaca		
	Micrurus frontalis	Antielapidico			
	Micrurus corallinus				
Syntex do Brasil S/A–Industria e Comercio Ciaxa Postal 951, São Paulo, Brasil	*Crotalus d. terrificus*		South American rattlesnake or cascabel		Pepsin digestion, and ammonium sulphate precipitation. Final solution contains 18% protein.
	Bothrops alternatus		Uruta		
	Bothrops atrox		Barba Amarilla		
	Bothrops jararaca		Jararaca		
	Bothrops jararacussu		Jararacussu		
	Bothrops cotiara		Cotiara		
Europe					
Institut Pasteur Annexe de Garches 92 (Hauts-de-Seine), Paris, France	*Vipera aspis*	Ipser V	Jura viper		Concentrated and purified to 12 to 13% protein
	Vipera berus		European viper		
	Vipera ammodytes	Ipser Europe	Long-nosed viper		
	Vipera aspis		Jura viper		
	Vipera berus		European viper		

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Europe					
	Bitis arietans	Bitis-Echis-Naja	Puff adder		
	Bitis gabonica		Gaboon viper		
	*Bitis nasicornis***		Rhinoceros viper		
	Echis carinatus		Saw-scaled viper		
	*Hemachatus haemachatus***		Ringhals		
	Naja haje		Egyptian cobra		
	Naja melanoleuca		Forest cobra		
	Naja nigricollis		Spitting cobra		
	*Naja nivea***		Cape cobra		
	Vipera ammodytes	Near and Middle East	Long-nosed viper		
	Vipera lebetina obtusa		Levantine viper		
	Vipera palestinae		Palestine viper		
	Cerastes cornutus		Horned viper		
	Cerastes vipera		Avicenna's viper		
	Echis carinatus		Saw-scaled viper		
	Naja naja		Indian cobra		
	Naja haje		Egyptian cobra		
	Naja naja kaouthia	Cobra	Yellow cobra		
	*Dendroaspis angusticeps***	Dendroaspis	Eastern green mamba		
	Dendroaspis jamesoni		Jameson's mamba		
	*Dendroaspis polylepis***		Black mamba		
	Dendroaspis viridis		Western green mamba		

(continues)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Europe					
Behring Institut Behringwerke AG D3550 Marburg/ Lahn Germany	*Vipera ammodytes* *Vipera berus*	*Europe*	*Long-nosed viper* *European viper*	*Vipera aspis* *Viper lebetina* *Vipera xanthina*	Prepared by pepsin digestion, and ammonium sulphate precipitation. Final solution contains 16% protein. Supplied in liquid form.
	Bitis lachesis *Bitis gabonica* *Echis carinatus* *Naja haje* *Vipera lebetina*	*North and West Africa*	*Puff adder* *Gaboon viper* *Saw-scaled viper* *Egyptian cobra* *Levantine viper*	*Cerastes cerastes* *Cerastes vipera* *Naja melanoleuca* *Naja nigricollis*	
	Bitis lachesis *Bitis gabonica* *Dendroaspis polylepis* *Naja haje*	*Central Africa*	*Puff adder* *Gaboon viper* *Black mamba* *Egyptian cobra*	*Bitis nasicornis* *Dendroaspis viridis* *Hemachatus haemachatus* *Naja melanoleuca* *Naja nigricollis*	
	Echis carinatus *Naja haje* *Vipera ammodytes* *Vipera lebetina*	Near and Middle East	Saw-scaled viper Egyptian cobra Long-nosed viper Levantine viper	*Cerastes cerastes* *Vipera xanthina* *Cerastes cornutus*	
Istituto Sieroterapico e Vaccinogeno Toscano "Sclavo," Via Fiorentine 1, Siena, Italy	*Vipera ammodytes* *Vipera aspis* *Vipera berus* *Vipera ursinii*	Antiviperin	Long-nosed viper Jura viper European viper Orsini's viper	All European vipers	Enzyme-refined and supplied in liquid form

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Europe					
Institute of Immunology Rockefellerova 2 Zagreb, Yugoslavia	*Vipera ammodytes*	Antiviperinum	Long-nosed viper	*Vipera berus* *Vipera aspis*	Solution digested with pepsin, and precipitated with ammonium sulphate
Institute of Epidemiology and Microbiology Sofia, Bulgaria	*Vipera ammodytes*		Long-nosed viper	*Vipera berus* *Vipera aspis*	Ammonium sulphate precipitation
Research Institute of Vaccine and Serum Ministry of Public Health Ul. Kafanova 93 Tashkent, U.S.S.R.	*Echis carinatus*	Monovalent Echis carinatus	Saw-scaled viper		No confirmation or recent letter indicating product or processing.
	Naja naja	Monovalent Naja naja	Indian cobra		
	Vipera lebetina	Monovalent Vipera lebetina	Levantine viper		
	Echis carinatus + *Naja naja*	Polyvalent Naja and Echis	Saw-scaled viper + Indian cobra		
	Naja naja + *Vipera lebetina*	Polyvalent Vipera and Naja	Indian cobra + Levantine viper		
Africa					
Institut Pasteur d'Algerie, Rue Docteur Laveran, Alger, Algeria	*Cerastes cerastes* + *Vipera lebetina*	Antiviperin	Horned viper + Levantine viper		Solution digested with pepsin and precipitated with ammonium sulphate

(continues)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Africa					
The South African Institute for Medical Research P.O. Box 1038 Johannesburg 2000, Republic of South Africa	*Hemachatus haemachatus* *Naja nivea* *Naja haje* *Naja melanoleuca* *Naja nigricollis* *Dendroaspis angusticeps* *Dendroaspis jamesoni* *Dendroaspis polylepis* *Bitis arietans* *Bitis gabonica*	Polyvalent	Ringhals Cape cobra Egyptian cobra Forest cobra Spitting cobra Eastern green mamba Jameson's mamba Black mamba Puff adder Gaboon viper	*Naja naja* *Ophiophagus hannah* *Pseudohaje goldii* *Walterinnesia egyptia* *Dendroaspis viridis*	Digested with pepsin and precipitated with ammonium sulphate
	Echis carinatus	Echis	Saw-scaled viper	*Echis coloratus* *Cerastes cerastes* *Cerastes vipera*	
	Dispholidus typus	Boomslang	Boomslang		
FitzSimmon's Snake Park, P.O. Box 1 Snell Parade, Durban, Republic of South Africa	*Dendroaspis angusticeps* *Dendroaspis jamesoni* *Dendroaspis polylepis*		Eastern green mamba Jameson's mamba Black mamba	*Dendroaspis viridis*	Digested with pepsin, precipitated with ammonium sulphate, and dialyzed
	Hemachatus haemachatus *Naja nivea* *Bitis arietans* *Bitis gabonica*		Ringhals Yellow cobra Puff adder Gaboon viper	*Naja naja* and African cobras	
Asia					
Central Research Institute Kasauli (Simla Hills), (H.P.) India	*Bungarus caeruleus* *Naja naja* *Vipera russelli* *Echis carinatus*	Polyvalent	Indian krait Indian cobra Russell's viper Saw-scaled viper	*Bungarus fasciatus* *Naja hannah*	Enzyme-refined, equine globulin supplied in liquid and lyophilized forms

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Asia					
Haffkine Bio-Pharmaceutical Corporation Ltd. Parel, Bombay, India	*Bungarus caeruleus*	Bungarus	Indian krait		Digested with pepsin, concentrated and lyophilized
	Naja naja	Naja	Indian cobra		
	Vipera russelli	Vipera	Russell's viper		
	Echis carinatus	Echis	Saw-scaled viper		
	Bungarus caeruleus	Polyvalent	Indian krait		
	Naja naja		Indian cobra		
	Echis carinatus		Saw-scaled viper		
Perusahaan Negara Bio Farma 9 Jalan Pasteur, Bandung, Indonesia	*Agkistrodon rhodostoma*		Malayan pit viper		Purified serum supplied in liquid form
	Bungarus fasciatus		Banded krait		
	Naja naja sputatrix		Malayan cobra		
Institut d'Etat des Serum et Vaccins Razi P.O. Box 656, Teheran, Iran	*Naja naja oxiana*		Oxus cobra		Prepared by pepsin digestion, and ammonium sulphate precipitation
	Vipera lebetina		Levantine viper		
	Echis carinatus		Saw-scaled viper		
	Pseudocerastes persicus		Persian horned viper		
	Vipera latasti		Snub-nosed viper		
	Agkistrodon halys		Mamushi		

(continues)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Asia					
	Naja naja oxiana *Vipera lebetina* *Vipera xanthina* *Echis carinatus* *Pseudocerastes persicus* *Agkistrodon halys*	Polyvalent	Oxus cobra Levantine viper Near East viper Saw-scaled viper Persian horned viper Mamushi	*Cerastes cerastes* *Eristicophis macmahonii* *Vipera aspis* *Vipera cerastes* *Vipera latasti* *Vipera x. palaestinae*	
Rogoff Medical Research Institute Beilinson Medical Center, Tel-Aviv, Israel	*Echis coloratus* *Vipera xanthina palaestinae*	Arabian Echis Palestine viper	Arabian saw-scaled viper Palestine viper		Whole venom plus resin-bound "neurotoxin" used as antigen Supplied as globulin fraction of horse serum in liquid form
Laboratory of Chemo-therapy and Serum Therapy 1 Furukyo-machi Kumamoto City, Kyushu, Japan	*Trimeresurus flavoviridis* *Agkistrodon halys*	Habu Mamushi	Habu Mamushi		Pepsin digestion, ammonium sulphate precipitation, and lyophilized
The Takeda Chemical Industries, Ltd. Higashi-Ku Osaka, Japan	*Trimeresurus flavoviridis* *Agkistrodon halys*	Habu Mamushi	Habu Mamushi		Pepsin digestion, ammonium sulphate precipitation, and lyophilized

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Asia					
Serum and Vaccine Laboratories Alabang, Mutinlupa, Rizal, Philippines	*Naja naja philippinensis*	Cobra	Philippine cobra		Concentrated and purified
National Institute of Preventive Medicine 161 Kun-Yang St., Nan-Kang, Taipei, Taiwan	*Agkistrodon acutus*	Agkistrodon	Long-nosed pit viper	*Trimeresurus mucro-squamatus*	Immunized with formalin—toxoid venom. Venom ammonium sulphate precipitated, and supplied in liquid or lyophilized form
	Bungarus multicinctus	Bungarus	Many-banded krait		
	Naja naja atra	Naja	Chinese cobra		
	Trimeresurus stejnegeri	Trimeresurus	Bamboo viper	*Agkistrodon acutus*	
	Trimeresurus mucro-squamatus	Trimeresurus	Chinese habu		
	Bungarus multicinctus	Naja-Bungarus	Many-banded krait		
	Naja naja atra	Naja-Bungarus	Chinese cobra		
Queen Saovabha Memorial Institute Rama 4 Road, Bangkok, Thailand	*Bungarus fasciatus*	Bungarus	Banded krait		Lyophilized whole serum
	Naja naja	Cobra	Indian cobra		
	Ophiophagus hannah	King cobra	King cobra		
	Vipera russelli	Russell's viper	Russell's viper		
	Agkistrodon rhodostoma	Malayan pit viper	Malayan pit viper		
	Trimeresuras albalabris and *T. erythrurus*	Bivalent	White-lipped tree viper		

(continues)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Australia					
Commonwealth Serum Laboratories†† 45 Poplar Road, Parkville, Victoria 3052, Australia	*Acanthophis antarcticus*	Death adder	Common death adder	*Acanthophis pyrrhus*	Prepared by pepsin digestion, and ammonium sulphate precipitation. The products are dialyzed and ultrafiltered to a final concentration of 17% protein.
	Notechis scutatus *Enhydrina schistosa*	Tiger-sea snake	Mainland tiger snake Beaked sea snake	*Austrelaps superba* *Pseudechis porphyriacus* *Tropidechis carinatus* Laboratory experiments indicated that antivenin neutralizes at least 12 different sea snake antivenins.	
	Oxyuranus scutellatus	Taipan	Taipan		
	Pseudonaja textilis, or	Eastern brown snake	Eastern brown snake	*Pseudonaja affinis* *Pseudonaja nuchalis*	
	Pseudechis australis	Brown snake	King brown snake	*Pseudechis australis* *Pseudechis porphyriacus*	
	Oxyuranus scutellatus *Acanthophis antarcticus* *Notechis scutatus* *Pseudechis australis* *Pseudonaja textilis*	Polyvalent (Australia–New Guinea)	Taipan Death adder Tiger snake King brown snake Eastern brown snake	*Austrelaps superba* *Pseudechis porphyriacus* *Pseudonaja affinis* *Pseudonaja nuchalis* *Pseudechis papuanus* *Parademansia microlepidota*	

Since the preparation of this table in March, 1979, the following data have been received, and supplement Table 8-2. I am indebted to doctors A. Ohsaka, Y. Sawai, F. Kornalik, and A. H. Mohamed for additional data. A revision of this table will be made by the W.H.O., Biologicals.

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	*Venoms Used in Preparation*	*Trade or Common Name*	*Common Name of Snake*	*Additional Venoms Neutralized**	*Comments*
Central and South America					
Instituto Nacional de Higiene Guayaquil Ecuador	*Bothrops atrox*	Anti-Bothrops	Barba Amarilla		Precipitated with ammonium sulphate and supplied in liquid form.
Instituto Nacional de Higiene Lima, Peru	*Bothrops atrox* *Bothrops brazili* *Lachesis muta*	Bothropico Polyvalent Anti-Laquesico	Barba Amarilla Bushmaster		Purified and lyophilized
Europe					
Institute for Sera and Vaccines W. Pieck Street Prague 2 C.S.S.R.	*Vipera ammodytes* *Vipera berus*	Venise	Long-nosed viper European viper	All European vipers	Pepsin digested and precipitated with ammonium sulphate. Supplied in liquid form.
Asia					
Industrial and Pharmaceutical Corporation Rangoon Burma	*Naja n. kaouthia* *Vipera r. siamensis*	Divalent	Siamese cobra Russell's viper		Precipitated with ammonium sulphate and lyophilized.
Serum Laboratory Shanghai Peoples Republic of China	*Agkistrodon halys* *Agkistrodon acutus*	Mamushi 100-Pace snake	Mamushi 100-Pace snake		Precipitated with ammonium sulphate and lyophilized.

(*continues*)

Table 8-2. Antivenins Available for the Treatment of Poisoning by Exotic Snakes (continued)

Producer or Distributor	Venoms Used in Preparation	Trade or Common Name	Common Name of Snake	Additional Venoms Neutralized*	Comments
Asia					
Research Institute for Microbial Diseases Ohsaka University Kita-ku Osaka Japan	*Trimeresurus flavoviridis* *Agkistrodon halys*	Habu Mamushi	Habu Mamushi		Pepsin digestion, and ammonium sulphate precipitation. Lyophilized.
Kitasato Institute Minato-ku Tokyo Japan	*Trimeresurus flavoviridis* *Agkistrodon halys*	Habu Mamushi	Habu Mamushi		Pepsin digestion and ammonium sulphate precipitation. Lyophilized.
Chiba Prefectural Serum Institute Ichikawa Chiba Prefecture Japan	*Trimeresurus flavoviridis* *Agkistrodon halys*	Habu Mamushi	Habu Mamushi		Pepsin digestion, and ammonium sulphate precipitation. Lyophilized.

Additional antivenins are prepared in Egypt (Al Agousa-Sharea Alvezara, Cairo): a polyvalent *Cerastes* serum and a polyvalent serum using *Naja haja, Cerastes cerastes,* and *Vipera cerastes* venoms. Specific data not available at time of writing.

*Additional venoms which said antivenin may neutralize, according to the producer. It can be expected that the antivenin will afford some protection, even though it might be slight, against the venoms of snakes of closely related species.
**Paraspecific.
†Data on antivenins from Japan supplied by Dr. A. Ohsaka, National Institute of Health, Tokyo, Japan.
††Manufacturer states that no true monospecific commercial antivenins are available. Horses are first "sensitized" to all major venoms and may then be used to produce a succession of separate antivenins.

References

1. Takahashi, W. Y., and Tu, A. T.: Puff adder snakebite. J.A.M.A., *211:*1857, 1970.
2. Rao, S. J.: Antivenin information and deposition centers. J.A.M.A., *212:*2123, 1970.
3. Russell, F. E.: Venomous bites and stings. *In* Berkow, R. (ed): The Merck Manual. ed. 13. Rahway, New Jersey, Merck, Sharp and Dohme Research Laboratories, 1977.
4. Busack, S. D.: Amphibians and Reptiles

Imported into the United States. Wildlife Leaflet 506, U.S. Fish and Wildlife Service, 1974.
5. Russell, F. E.: Clinical aspects of snake venom poisoning in North America. Toxicon, 7:33, 1969.
6. Jenkins, M., and Russell, F. E.: Physical therapy for snake venom poisoning. Phys. Ther., *54:*1298, 1974.
7. Parrish, H. M.: Ophidiasis. An unusual occupational hazard. Indust. Med. Surg., *27:*63, 1958.
8. Christy, N. P., *et al.*: Exotic snakebites in an urban setting: heparin therapy in a patient bitten by a saw-scaled viper (*Echis carinatus*). Trans. Am. Clin. Climatol. Assoc., *84:*37, 1973.
9. Minton, S. A.: Poisonous snakes: Parts I & II. Clin. Med., *85:*13, 1978.
10. Warrell, D. A., *et al.*: Poisoning by bites of the saw-scaled or carpet viper (*Echis carinatus*) in Nigeria. Q. J. Med., *46:*33, 1977.
11. Reid, H. A.: Prolonged defibrination syndrome after bite by the carpet viper *Echis carinatus*. Br. Med. J., *2:*1326, 1977.
12. Warrell, D. A., *et al.*: Poisoning by bites of the saw-scaled or carpet viper (*Echis carinatus*) in Nigeria. Q. J. Med., *45:*627, 1976.
13. Visser, J., and Chapman, D. S.: Snakes and Snakebite. Cape Town, Purnell & Sons, 1978.
14. Fitzsimons, V. F. M.: Snakes of Southern Africa. Johannesburg, Purnell & Sons, 1962.
15. Hobley, C. W.: The snakes of British East Africa. J. E. Afr. Uganda Nat. Hist. Soc., 3:43, 1912.
16. Scholefield, S. W. J.: On spitting cobras. J. E. Afr. Uganda Nat. Hist. Soc., *1:*61, 1912.
17. Loveridge, A.: Notes on snakes and snake-bites in East Africa. Bull. Antivenin Inst. Amer., *1:*106, 1928.
18. Corkhill, N. L.: Snake stories from Kordofan. Sudan Notes and Records, *18:*243, 1935.
19. Bogert, C. M.: Dentitional phenomena in cobras and other elapids with notes on adaptive modifications of fangs. Bull. Amer. Mus. Nat. His., *81:*285, 1943.
20. Ditmars, R. L.: Snakes of the World. New York, Macmillan, 1931.
21. Rose, W.: Veld and Vlei. An account of South African frogs, toads, lizards, snakes, and tortoises. Capetown, 1929.
22. Noyes, H.: Spitting cobras. Field, *164:*1477, 1934.
23. Warrell, D. A., *et al.*: Necrosis, haemorrhage and complement depletion following bites by the spitting cobra (*Naja nigricollis*). Q. J. Med., *45:*1, 1976.
24. Warrell, D. A., and Ormerod, L. D.: Snake venom ophthalmia and blindness caused by the spitting cobra (*Naja nigricollis*) in Nigeria. Am. J. Trop. Med. Hyg., *25:*525, 1976.
25. Pergola, A.: Allezioni oculari da veleno di *Naja nigricollis* (obsservazioni cliniche). Soc. It. Med. e Ig. Trop., Dec. 6, 80, 1941.
26. Sarnelli, T.: Manifestazion oculari dell'olidismo e, congiuntiviti da sputo di serpenti nelle nostre clonie. Archivo Italiano di Scienze Mediche Coloniali e di Parassitologia Fasc., *11:*769, 1935.
27. Reid, H. A.: Cobra-bites. Br. Med. J., *2:*540, 1964.
28. Moore, W. J.: A case of snakebite. Indian med. Gaz., *3:*103, 1868.
29. Bull, G. H.: Recovery after the bite of a cobra. Indian med. Gaz., *15:*271, 1880.
30. Hennessy, P. H.: A snakebite (cobra) case. Indian med. Gaz., *53:*154, 1918.
31. Banerjee, R. N., and Siddiqui, Z. A.: Epidemiological study of snake-bite in India. *In* Rosenberg, P. (ed.): Toxins, Animal, Plant and Microbial. Oxford, Pergamon Press, 1978.
32. Rosenfeld, G., Nahas, L., de Cillo, D. M., and Fleury, C. T.: *In* Cintra do Prado, F., Ramos, J., and Ribeiro do Valle, J. (eds.): Atualizacao Terapeutica. Rio de Janeiro, Livraria Luso-Espanhola e Brasileira, 1957.
33. Rosenfeld, G.: Unfalle durch Giftschlange. *In* Die Giftschlangen der Erde. Marburg, Behringwerke-Mitteilungen, 1963.
34. Rosenfeld, G.: Symptomatology, pathology, and treatment of snake bites in South America. *In* Bucherl, W., and Buckley, E. (ed.): Venomous Animals and Their Venoms. New York, Academic Press, 1971.
35. Worrell, E.: Dangerous Snakes of Australia and New Guinea. Sydney, Angus and Robertson, 1961.
36. Cogger, H.: Australian Reptiles in Colour. Honolulu, East-West Center Press, 1967.
37. Campbell, C. H.: The treatment of suspected venomous snake bite. Med. J. Aust., *2:*493, 1963.

38. Sutherland, S. K.: Treatment of snake bite in Australia. Some observations and recommendations. Med. J. Aust., *1*:30, 1975.
39. Hood, V. L., and Johnson, J. R.: Acute renal failure with myoglobinuria after snake bite. Med. J. Aust., *2*:638, 1975.
40. Harris, J. B., Johnson, M. A., and MacDonell, C.: Taipoxin, a presynaptically active neurotoxin, destroys mammalian skeletal muscle. Proc. Br. Pharmacol. Soc. Meeting, July, 1977.
41. Broad, A. J., Sutherland, S. K., and Coulter, A. R.: The lethality in mice of the dangerous Australian snake venoms and certain exotic snake venoms. Toxicon (In press).
42. Campbell, C. H.: Clinical aspects of snake bite in the Pacific area. Toxicon, *7*:25, 1969.
43. Sutherland, S. K.: Venomous Australian creatures: the action of their toxins and the care of the envenomated patient. Anaesth. Intens. Care, *2*:316, 1974.
44. Sutherland, S. K.: Treatment of venomous animal bites and stings in Australia. Med. J. Aust., *1*:177, 1976.
45. Russell, F. E., and Lauritzen, L.: Antivenins. Trans. Roy. Soc. Trop. Med. Hyg., *60*:797, 1966.